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ORAL SEPSIS
IN ITS RELATIONSHIP TO
SYSTEMIC DISEASE

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SYSTEMIC DISEASE

BY

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TO ST. MARGARET'S HOSPITAL, KANSAS
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TO MY FATHER
HENRY BUFORD DUKE

THIS BOOK
IS AFFECTIONATELY
DEDICATED

PREFACE

In the preparation of this little book, the aim has been to present as briefly and clearly as possible the rather complex relationship which frequently exists between infections of the gum and alveolar process and certain systemic disorders. This has necessarily involved a discussion of some of the more complex problems of bacteriology, immunology, and pathology as well as of dentistry. In so small a space it has not been possible to take up any of these subjects in great detail, and for this reason those especially interested in certain particular phases are referred to the writings of Rosenow, Billings, Schottmüller, Poynton and Payne, Libman, Vaughan, von Pirquet, Wolff-Eisner, Richet, Rosenau and Anderson, and other investigators to whom we are indebted for many of our more recent ideas which have a bearing upon this very important subject.

In the opinion of the writer, the discovery of the relationship which frequently exists between the various chronic infections and systemic disease marks a great practical advance in the science and therapy of medicine. For centuries it has been evident to physicians that certain acute inflammatory lesions give rise to systemic disturbances. One of the main advances of recent years, therefore, has been the disclosure of the fact that small and apparently innocent infections which give rise to little or no local disturbance may likewise be the source of serious generalized disease.

For the proper care of medical cases, the hearty and intelligent cooperation of the dentists is absolutely essential. In the writer's experience, it has been found

that the teeth should be taken into account in nearly every medical case. For this reason, the welfare of the affected individual demands that the dental surgeon understand and appreciate the problems and aims of the physician, and that he be willing at times to sacrifice apparently useful teeth for the sake of the patient's general welfare. Consequently, the main purpose of this volume has been to assemble facts which show, first of all, that dental sepsis is an extremely common condition, and, second, that it may cause serious systemic disorder in many different ways.

For the sake of completeness, it has been necessary to discuss some of the purely dental problems. These have been discussed purely from a pathologic viewpoint based upon an experience with more than one thousand carefully studied medical cases observed in a consulting office practice on whom dental roentgenograms were made as part of a routine examination. In all over eight thousand dental films were taken. It is believed and sincerely hoped that the views here expressed are in harmony with those of the dental surgeons who have given the problem of oral sepsis the attention and study it deserves. For a more detailed account of this subject from a dental standpoint, the reader is referred to the publications of the investigators in this field: G. V. Black, and A. D. Black, Hartzell, Price, Rhein, Thoma, Grieves, etc.

Finally, I wish to acknowledge with thanks the hearty cooperation of my friend and coworker in this study, Rex Dively, under whose direction the roentgenologic work was done, to L. S. Milne and W. A. Myers, my office associates, to Joseph Kelly and Miss Rose Mary Trott, for their careful work in clinical pathology, and to Miss Elizabeth Leas, assistant in the roentgenologic laboratory.

WILLIAM W. DUKE.

Kansas City, Mo.

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ORAL SEPSIS IN ITS RELATIONSHIP TO SYSTEMIC DISEASE

CHAPTER I

INTRODUCTION

The discovery of a relationship between ill health and defective teeth is by no means recent. It has received casual mention in the older literature and has been independently recognized, perhaps for centuries, by practitioners of medicine and dentistry. The subject has not received the prominence it deserves, however, previous to the past decade. We wish to quote *in toto* an article published by Benjamin Rush, one of the signers of the Declaration of Independence, and one of America's most noted physicians, on observations commenced by him in 1801. This remarkable article, written before the discovery of bacteria and when our knowledge of pathology was meager indeed, harmonizes in its essentials with the more popular views of the present day. I have taken the liberty of italicizing several striking sentences.†

MEDICAL INQUIRIES AND OBSERVATIONS*

By Benjamin Rush, M.D.

Professor of the Institute and Practice of Medicine and Clinical Practice in the University of Pennsylvania

“Some time in the month of October, 1801, I attended Miss A. C. with rheumatism in her hip joint, which yielded for a while, to the several remedies for that dis-

*Vol. I, p. 199, published in 1818 by M. Carey & Son, Philadelphia. This article was found by Mrs. Rose M. Hibbard of the Kansas City Medical Library in the private collection of Dr. A. E. Hertzler. It has also been referred to by George N. Kreider, A. D. Black, and David Riesman.

ease. In the month of November it returned with great violence, accompanied with a severe toothache. Suspecting the rheumatic affection was excited by the pain in her tooth, which was decayed, I directed it to be extracted. The rheumatism immediately left her hip, and she recovered in a few days. She has continued ever since to be free from it.

“Soon after this I was consulted by Mrs. J. R. who had been affected for several weeks with dyspepsia and toothache. Her tooth, though no mark of decay appeared in it, was drawn by my advice. The next day she was relieved from her distressing stomach complaint, and has continued ever since to enjoy good health. *From the soundness of the external part of the tooth, and the adjoining gum, there was no reason to suspect a discharge of matter from it had produced the disease in her stomach.*

“Some time in the year 1801 I was consulted by the father of a young gentleman in Baltimore, who had been affected with epilepsy. I inquired into the state of his teeth, and was informed that several of them in his upper jaw were decayed. I directed them to be extracted, and advised him after to lose a few ounces of blood, at any time when he felt the premonitory symptoms of a recurrence of his fits. He followed my advice, in consequence of which I had lately the pleasure of hearing from his brother that he was perfectly cured.

“*I have been made happy by discovering that I have only added to the observations of other physicians, in pointing out a connection between the extraction of decayed and diseased teeth and the cure of general diseases.* Several cases of efficacy of that remedy in relieving headache and vertigo are mentioned by Dr. Darwin. Dr. Gater relates that Mr. Petit, a celebrated French surgeon, had often cured intermitting fevers, which had resisted the bark for months, and even years, by this prescription; and he quotes from his works two cases, the one of

consumption, the other of vertigo, both of long continuance, which were suddenly cured by the extraction of two decayed teeth in the former, and of two supernumerary teeth in the latter case.

“In the second number of a late work, entitled *Bibliothèque Germanique Medico Chirurgicale*, published in Paris, by Dr. Bluver and Dr. Delaroche, there is an account, by Dr. Siebold, of a young woman who had been affected for several months with great inflammation, pain, and ulcers, in her right upper and lower jaws, at the usual time of the appearance of the catamenia, which at that period were always deficient in quantity. Upon inspecting the seat of those morbid affections, the doctor discovered several of the molars in both jaws to be decayed. He directed them to be drawn, in consequence of which the woman was relieved of the monthly disease in her mouth, and afterwards had a regular discharge of her catamenia.

“*These facts, though but little attended to, should not surprise us, when we recollect how often the most distressing general diseases are brought on by very considerable inlets of morbid excitement into the system. A small tumor, concealed in the fleshy part of the leg, has been known to bring on epilepsy. A trifling wound with a splinter or a nail, even after it has healed, has often produced a fatal tetanus. Worms in the bowels have produced internal dropsy of the brain, and a stone in the kidney has excited the most violent commotions in every part of the system. Many hundred facts of a similar nature are to be met with in the records of medicine.*

• “*When we consider how often the teeth, when decayed, are exposed to irritation from hot and cold drinks and aliments, from pressure by mastication, and from the cold air, and how intimate the connection of the mouth is with the whole system, I am disposed to believe they are often the unsuspected causes of general, and par-*

ticularly of nervous diseases. When we add to the list of those diseases the morbid effects of the acrid and putrid matters, which are sometimes discharged from the carious teeth, or from the ulcers in the gums created by them, also the influence which both have in preventing perfect mastication, and the connection of that animal function with good health, *I can not help thinking that our success in the treatment of all chronic diseases would be very much promoted, by directing our inquiries into the state of the teeth in sick people, and by advising their extraction in every case in which they are decayed. It is not necessary that they should be attended with pain, in order to produce diseases,* for splinters, tumors, and other irritants before mentioned, often bring on disease and death, when they give no pain, and are unsuspected causes of them. This translation of sensation and motion into parts remote from the place where impressions are made, appears in many instances, and seems to depend upon an original law of the animal economy.”

The following generalizations are recognized by many who have interested themselves in the bearing which the teeth have upon health. Very few men of fifty years or over who show advanced stages of dental sepsis are normal physically. The vast majority have chronic disease. There are, of course, notable exceptions to this. Conversely, adults of fifty or over who have perfect teeth free from sepsis are often remarkably free from chronic disease (inflammatory in origin) and are usually well preserved. Striking examples of individuals seventy to ninety years of age with nearly perfect teeth and splendid health impress one with the reality of a relationship between the two. Men of advanced age who have worn false teeth for a number of years have better health as

a rule than the average of their age who have retained defective teeth.

• In earlier years the relationship between ill health and defective teeth was attributed to caries. It was very apparent that this rendered teeth unfit for mastication and that the lack of this function might be a source of digestive disturbance. Infection of food by carious teeth during the process of chewing seemed a factor as also did the swallowing of putrid matter from carious teeth and of pus from infected gums.

It is not unlikely that the swallowing of poorly masticated food, the swallowing of pus, putrid material, etc., has some slight untoward influence upon digestion. It does not seem probable, however, that it has an important bearing upon the many systemic ills for which the teeth are now thought to be remotely responsible. The fact is that the mucous membrane of the gastrointestinal tract when in healthy condition can tolerate a great deal of abuse. It can tolerate poorly masticated food, and, as a rule, can destroy septic material when swallowed. The serious effect of oral sepsis is rarely through this channel in individuals who are otherwise normal.

Pyorrhea is mentioned as a possible factor in the etiology of anemias, joint troubles, etc., in some of the older textbooks. Its importance, however, as a frequent source of chronic infection distributed by the blood to remote organs is a recent suggestion. The discovery of the frequency of alveolar abscesses and granulomata and their importance as a source of ill health is of still more recent date and has been brought to light through dental roentgenology. The fact that alveolar abscesses are of greater pathologic import, so far as general health is concerned, than pyorrhea seems at once evident. Alveolar abscesses lack drainage. In pyorrhea drainage is often good. Not only are alveolar abscesses blind, but their localization in bony tissue renders them a more

serious detriment to health than septic foci of equal size in soft tissues such as the gums where expansion is possible. Apical sepsis would appear a greater factor than pyorrhea in the causation of disease for another important reason; namely, pyorrhea gives rise to symptoms which usually attract the attention of both patient and dentist. Alveolar abscesses do not give rise, as a rule, to a single disagreeable symptom by which either patient or dentist is led to suspect their existence. Consequently, many individuals have been subject to the effect of sepsis in the alveolar process for years even though they have paid scrupulous attention to their teeth and visited the dentist frequently. The frequent failure of alveolar abscesses to cause pain or other local evidence of trouble often makes it difficult to convince patients that their teeth need attention; in fact, dentists themselves occasionally refuse to believe that certain teeth are diseased, even when the fact can be clearly demonstrated in dental roentgenograms. This frequently leads to controversies and unfortunate situations.

The incidence of infection in the gums and alveolar process is astonishingly great. It is not frequently found in individuals of less than twenty years, but is found in the vast majority of adults. It is found oftener and in greater extent as age advances, and in late adult life few escape it entirely.

One of the greatest sources of dental sepsis aside from decay, tartar, and gross neglect, is dental work. It is not altogether fair to the dentists to say defective dental work, for the same is very frequently true even of dental work carried out according to the more approved methods of the past decade. As a broad, general rule it can be said of individuals whose teeth have received careful attention that the amount of oral sepsis at a given age varies largely with the amount of dental work and that few individuals with a considerable amount of

dental work are free from it. It is generally stated by dentists who have interested themselves in the septic conditions of the mouth that fifty per cent or more of devitalized teeth show roentgen shadows at the root apices which are indicative of chronic sepsis and that a very large proportion of crowns, fillings, and bridges project at the gum margin enough to irritate the gum and leave pockets in which food material can lodge and putrify and lead almost inevitably to pyorrhea.

Dentists have been wonderfully successful in preserving the visible portion of the teeth and in making restorations which look well and serve well for purposes of mastication, but very few have given the attention to sepsis which it now appears to deserve. Fillings, crowns, and bridges have not been constructed in the past with a paramount purpose of avoiding sepsis, and have often been attached to teeth so badly infected that a sanitary result could not have been hoped for. The sins of commission have been no greater than the sins of omission, for many of the most skilled dentists even at the present day allow patients to leave their offices looking lightly upon a degree of sepsis which may be not only detrimental to their physical welfare, but may also lead eventually to the loss of more teeth. In mentioning this, we do not wish to question the skill of dentists, but rather to emphasize the fact that unanimity of opinion between physicians and dentists concerning the diagnosis of dental sepsis and concerning the remote effect of the same is urgently needed, as is also hearty cooperation in its treatment. In the past the practice of dentistry has been directed toward the preservation of the teeth almost solely for mechanical and cosmetic purposes. In the future it appears, it is destined to have as one of its chief aims the prevention and cure of disease.

CHAPTER II

PYORRHEA ALVEOLARIS

Pyorrhea alveolaris is rarely due to one cause alone. In an overwhelming majority of cases it is due to one chief cause and several important contributing causes. The more important causes are mechanical conditions which expose the gums repeatedly to injury or to the irritating effects of putrefying food material, secretions, etc.; systemic or local conditions which increase the susceptibility of the gums to infection and finally infection itself. The successful treatment of pyorrhea depends upon the removal of each abnormal condition which takes part in its etiology.

Causes of Pyorrhea Alveolaris

1. *Microorganisms*; streptococcus group, pneumococcus group, staphylococcus group, amebæ, spirochetæ, and fusiform bacilli of Vincent's angina, bacilli of many types, putrefactive organisms, etc.

2. *Chronic irritation or trauma*, such as that caused by tartar, decay, malocclusion, defective dental work, and, perhaps, in rare instances, by the improper use of toothpicks, toothbrushes, etc.

3. *Unsanitary conditions*, such as may be caused by pockets and irregularities of the teeth which make possible the lodgement and putrefaction of secretions, food materials, etc. Such may occur as a result of defective dental work, tartar, decay, irregularity of the teeth, etc.

4. *Conditions which prevent the normal massage of the gums and cleaning of the teeth* by the excursion of food, the tongue, and cheeks during the process of mastication;

namely, malocclusion, irregularity of the teeth, and defective dental work.

. 5. *Constitutional conditions which increase the susceptibility of the gums to infection*; namely, diabetes, pregnancy, lactation, alcoholism, lead poisoning, the use of mercury and potassium iodide in therapy, chronic debilitating diseases, blood diseases, anemia, etc., diseases of the ductless glands, scurvy, acute infectious diseases,

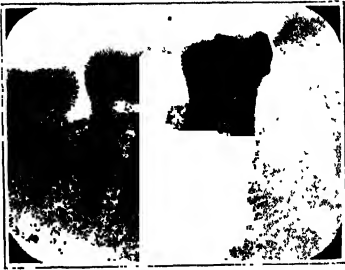


Fig. 1.



Fig. 2.



Fig. 3.

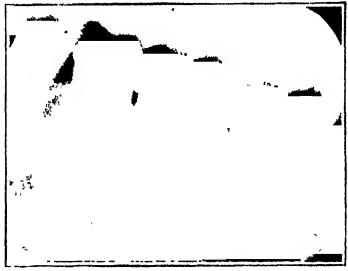


Fig. 4.

Figs. 1-4.—Illustrations showing a normal condition of roots and alveolar process. Figs. 1 and 2, in adult life. Figs. 3 and 4, before eruption of second teeth.

chronic infections, such as infected tonsils, alveolar abscesses, infected nasal sinuses, chronic appendicitis, cholecystitis, etc.

The above factors vary in their relative importance, and each factor varies in its degree of importance. Several factors play a part in the majority of cases. When this is the case, relatively unimportant factors may play important roles. For example, acute trauma, such as

that brought about by injury or by the use of toothpicks, stiff toothbrushes, etc., seldom or never causes pyorrhea in normal individuals with normal teeth, in fact, stiff brushes are used in prophylaxis against pyorrhea. In a patient with diabetes, however, or in pregnant women, acute trauma may initiate an infection of the gums, especially if, in addition to this, the patient has tartar or defective dental work. Likewise, the irritating effect of defective dental work which might be well tolerated by a normal individual might be a source of severe pyorrhea in an individual taking intensive doses of mercury or potassium iodide, or in an individual with irregular teeth having accumulations of tartar, etc.

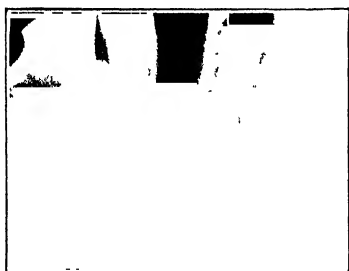


Fig. 5.

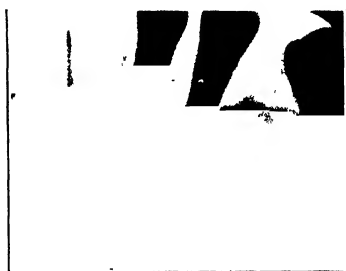


Fig. 6.

Figs. 5 and 6.—Illustration showing erosion of the alveolar process (moderate in degree) due to chronic infection derived from the gum margin.

As previously mentioned, the majority of individuals with pyorrhea show the presence of two or more causative factors. Unfortunately, in many instances, numerous factors can be found. The reason for this becomes apparent if the causative factors are traced to their sources of origin. For example, enlarged tonsils and adenoids are common causes of mouth-breathing in children. Mouth-breathing is a common cause of malocclusion and irregularity of the teeth. Malocclusion interferes with the self-cleansing of the teeth, prevents the normal massage of the gums, and allows the accumulation of tartar. The unsanitary conditions caused there-

by predispose to decay and make dental work necessary. Decay, tartar, and dental work are the most frequent causes of pyorrhea. Individuals who have been mouth-breathers by reason of hypertrophied tonsils and adenoids are likely, therefore, to have, not only defective teeth which are prone to cause pyorrhea, but also hypertrophied infected tonsils which may lower resistance to infection and which in this way increase the susceptibility



Fig. 7.



Fig. 8.



Fig. 9.



Fig. 10.

Figs. 7-10.—Case of pyorrhea alveolaris of long standing. Shows great destruction of the alveolar process.

of the gums to infection. Such individuals are also likely to have one or more systemic diseases as a result of acute or chronic tonsillitis, which may cause an additional lowering of resistance. All of the above factors singly or combined may play important parts in the pathogenesis of pyorrhea. In the average medical case, not one, but several predisposing factors can usually be found.

Infection is seldom or never a sole cause of pyorrhea.

Pyorrhea would seem theoretically impossible, however, without infection as a primary cause. Malocclusion, defective dental work, etc., might cause pressure atrophy of the gum and alveolar process, but could never cause the chronic inflammatory changes of pyorrhea unless infection were superimposed.

The *microorganisms* found in septic pockets about the



Fig. 11.



Fig. 12.



Fig. 13.



Fig. 14.

Figs. 11-14.—Illustrations of cases in which the alveolar process has been so destroyed by infection that some of the teeth are held in place by soft tissues alone. Pressure on such teeth during mastication forces them down on cushions of chronically infected tissue. They would appear a greater menace to health than infected teeth whose roots are still embedded in bone and held in place more firmly. The teeth shown in Figs. 13 and 14 had been treated for two years by a pyorrhea specialist. The surface of the gum was pink and appeared relatively healthy. Marked relief of systemic complaint followed the extraction of teeth in each of the cases illustrated above.

teeth are numerous and varied. If the superficial pus in pyorrhea pockets is removed and examined, a great variety are found coexisting; namely, streptococci, pneumococci, diplococci, staphylococci, bacilli, spirochetes, and amebæ, of various sizes and strains. If the superficial

pus is wiped away and a culture or smear is taken from the deeper areas, the members of the streptococcus and staphylococcus group are found most constantly. Streptococci usually predominate in numbers and are often in relatively pure culture. *Streptococcus viridans*, *streptococcus hemolyticus*, and amebæ can be demonstrated in

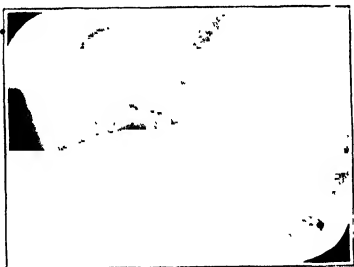


Fig. 15.



Fig. 16.

Figs. 15-16.—Pyorrheal abscesses. These teeth are held more firmly in place by the remains of the alveolar process than those shown in illustrations 11 to 14.

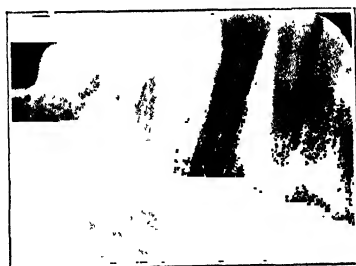


Fig. 17.

Fig. 17.—Abscess at the root apex of a vital tooth. Tooth responded to all tests for vitality of pulp. The infection was derived from the gum margin. The anterior table of the alveolar process had been eroded as far down as the apex of the root. The gum, however, appeared relatively healthy on casual examination. The tooth had never caused pain or other symptoms which had attracted the attention of the patient.

almost every case, and a few colonies of staphylococcus aureus and albus in the majority of cases.

It seems improbable that any individual organism plays a specific role in the pathology of the disease. Many of the organisms which can be isolated are pathogenic and capable of causing acute or chronic inflammatory change in many tissues under favorable conditions. One doubts, however, if any of them ever gain

a foothold in the tissues of a normal healthy gum, and cause local disease unless the resistance of the gum against infection is lowered by some coexisting abnormal condition. In fact, one might be justified in believing that if a pure culture of bacteria obtained from a case of pyorrhea were applied directly to the gum, the bacteria would probably be washed away and killed by the secretions of the mouth, and do no harm unless the gum



Fig. 18.

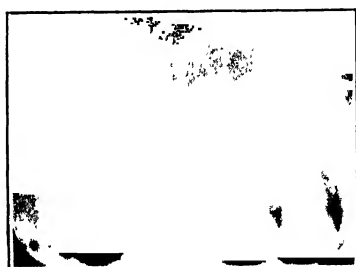


Fig. 19.

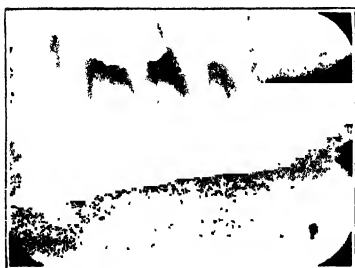


Fig. 20.



Fig. 21.

Figs. 18-21.—Illustrate the ill effect upon the alveolar process of careless dental work.

had been previously rendered susceptible to attack by local injury, lowered resistance, or both combined.

A spirillum associated with a fusiform bacillus found in great numbers in Vincent's angina is perhaps worthy of special mention. These organisms are found in small numbers in pus expressed from pockets about the teeth and tonsils in normal individuals. They are occasionally found in overwhelming numbers and in almost pure culture in cases of severe rapidly advancing pyorrhea. This

type of disease may progress with great rapidity, and may cause rapid destruction of the soft parts and alveolar process even in patients with relatively normal teeth and gums. It is more commonly observed, however, in individuals in whom irregularity of teeth, tartar, defective dental work, or careless habits render the mouth unsanitary. This disease usually yields rapidly to local treatment.

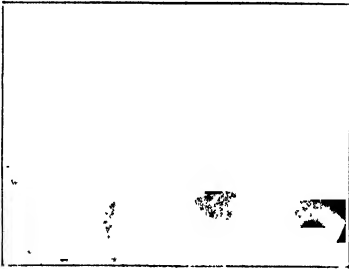


Fig. 22.



Fig. 23.



Fig. 24.

Figs. 22-24.—Illustrate the ill effect upon the alveolar process of careless dental work.

The presence of amebæ can be demonstrated in the vast majority of chronic lesions of the gum. They are often present in great number, especially in the deeper pockets. This discovery led Bass and Johns to suggest the use of emetine in the treatment of pyorrhea. It is difficult to determine whether or not amebæ play an important role in the pathology of pyorrhea or whether they are harmless secondary invaders. It appears true, however, that emetine has a certain limited sphere of useful-

ness in therapy. Its effect is not permanent, however, unless the mechanical and sanitary condition of the mouth is properly cared for.

Temporary trauma, such as might be caused by an acute injury or by the use of toothpicks or toothbrushes and by the irritating effects of tobacco, is perhaps never in itself a cause of pyorrhea in healthy individuals with regular, clean teeth. Temporary trauma may be a contributing cause of minor importance, however, in patients with unsanitary oral cavities.

Chronic irritation and repeated trauma of the gum, and unsanitary conditions due to tartar, decay at the gum margin, poorly constructed fillings, crowns and bridges, malocclusion, irregularity of the teeth, etc., are among the most important and most frequently observed causes of pyorrhea alveolaris. If teeth were all regular and kept clean, and if dental work was always properly constructed, pyorrhea would be a relatively rare disease.

Pyorrhea due to the above causes is likely to occur in localized areas of infection at first. It is found frequently under defective fillings, crowns and bridges, and in such locations there may be extensive destruction of both gum and alveolar process. Later the infection is likely to become more general. It hardly comes within the scope of a medical man to say more concerning this purely dental problem.

Lowered resistance to infection has an important bearing upon the development of pyorrhea. Dentists who neglect this factor are likely to have a number of failures in their efforts to cure pyorrhea. Resistance may be lowered by the following diseases to such an extent as to render the gums abnormally susceptible to infection:

First, by constitutional conditions, such as diabetes, pregnancy, lactation, alcoholism, debilitating diseases, blood diseases, anemia, diseases of the ductless glands, lead poisoning, and scurvy, and by the therapeutic use

of mercury and potassium iodide. It is well known that pyorrhea frequently starts and advances rapidly during pregnancy and lactation, especially in individuals with

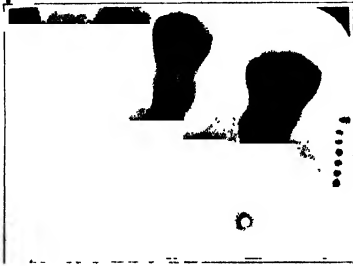


Fig. 25.



Fig. 26.

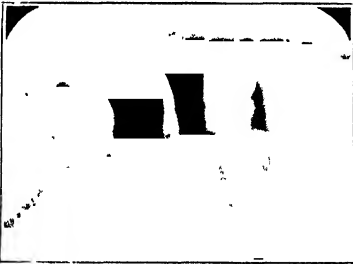


Fig. 27.



Fig. 28.



Fig. 29.

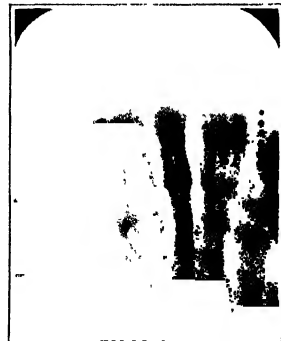


Fig. 30.

Figs. 25-30.—Illustrations show deposits on necks of roots. In some instances there appears to be bone proliferation of the root due to chronic irritation of the periodontal membrane just below the gum margin.

defective and unsanitary oral cavities; also that pyor-rheal sepsis, like other forms of sepsis, is likely to occur

in patients with diabetes, anemia, etc. The reason is that the defensive mechanism in patients with these conditions is definitely reduced in its power to combat infection so that they become fit subjects for pneumonia, tuberculosis, furunculosis, pyorrhea, and other infections. Such patients may combat infection normally if the systemic condition is relieved. A case of acute superficial pyorrhea observed in a patient with diabetes who had regular, normal teeth, cleared up without local treatment a few days after the urine was rendered free from sugar.

Second, resistance against infection may be lowered by infection. It may be lowered by the more localized infections, such as tonsillitis, alveolar abscesses, infected nasal sinuses, cholecystitis, appendicitis, etc., and by the more generalized infections such as typhoid fever. The bearing which infection in distant organs may have upon the development and course of pyorrhea is important and often striking. Pyorrhea is sometimes noticed first or apparently cured cases recur soon after an attack of tonsillitis, or after the development of an acute alveolar abscess, or after a sinus or gall-bladder infection. This is especially true of individuals with unsanitary mouths. It may be attributed to the fact that resistance is lowered by the acute infection to such an extent that the organisms about the teeth flourish, invade the tissue of the gum and cause local inflammation. Analogous examples of exacerbation or recurrence of apparently healed inflammatory processes after the development of acute infections are commonly met with in the practice of medicine. It is a common occurrence, for example, for latent tuberculosis to become active after an attack of bronchitis or acute tonsillitis. Chronic appendicitis, cholecystitis, or a chronic latent Neisser infection may undergo an acute exacerbation during or following acute bronchitis or tonsillitis. Urethritis that has been clini-

cally well for months has been known to recur after an attack of la grippe. A healing furuncle may discharge more pus or the scars of recently healed furuncles may



Fig. 31.



Fig. 32.



Fig. 33.



Fig. 34.



Fig. 35.

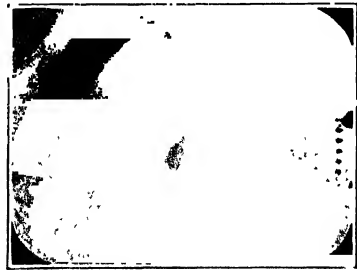


Fig. 36.

Figs. 31-36.—Case of chronic recurrent pyorrhea alveolaris in a patient, age 70, with remarkably regular, normal, clean teeth. This case did not yield well to treatment for pyorrhea until after the extraction of the abscessed tooth shown in Fig. 32. It seemed to be a case in which the resistance of the gum against infection was lowered by the undiscovered untreated abscessed tooth. The systemic condition of the patient was very much improved after the extraction of the abscessed tooth and the condition of the gum was kept normal thereafter with relatively little care.

itch, become red and even discharge pus after the development of a fresh furuncle or after an attack of tonsillitis. A patient we observed who had specific disease

which had been latent for four years had an extensive papillary brown-red rash and gave a positive Wassermann test one week after recovery from typhoid fever.

The above examples are mentioned to illustrate the fact that resistance against infection may be lowered by infection. It may be lowered either by focal infections or by the more widespread infections. For this reason, alveolar abscesses, chronically infected tonsils or adenoids, infected nasal sinuses, chronic appendicitis, chronic cholecystitis, and other chronic infections may be insurmountable obstacles to the permanent cure of pyorrhea by local treatment alone. The gums may improve and remain healthy so long as the teeth are kept scrupulously clean and free from tartar, but so soon as the hygiene of the mouth is neglected and resistance is lowered by fatigue, exposure to cold, indulgence in alcohol, etc., increased activity of some chronic infection may cause a further lowering of resistance and the lighting up of a latent infection of the gums. Chronic foci of infection are frequently contributing causes of pyorrhea and frequently render its permanent cure difficult.

CHAPTER III

ALVEOLAR ABSCESSSES

Alveolar abscesses have two sources of origin: one, through infection of the alveolar process from the root canal of teeth with infected pulp; the other through infection from the gum margin after pyorrheal erosion of the alveolar process. The former type of abscess is the more common.

The occurrence of alveolar abscesses is astonishingly great. Black in a recent article reports the finding of abscesses at the roots of forty-seven per cent of devitalized teeth. His observations were made on individuals without reference to complaint concerning mouth conditions or state of health, and he thinks represents the average for persons of less than forty years of age. In examining one thousand medical cases in office practice on whom dental films were made as a routine procedure, we found areas of bone absorption at the roots of eighty-one per cent of all nonvital teeth. The majority of the patients were over forty years of age.

Considerable variation in statistics concerning abscessed teeth obtained by different observers can be expected. The relative number of infections would appear to vary with the class, age, and complaint of the patients examined. The number diagnosed vary because of slight differences of opinion concerning the interpretation of dental roentgenograms.* If the slightest evidence of bone absorption at the apex of a devitalized tooth is considered evidence of sepsis, then the number of positive findings is great, indeed. Of the total number of de-

*In these statistics teeth were counted as abscessed only when the roentgen shadows had such an appearance as would leave relatively little doubt in the mind of the average careful observer concerning the diagnosis.

vitalized teeth examined by Diveley and myself, the area of absorption was extremely slight in thirty-two per cent, so slight in fact that opinions would differ as to whether

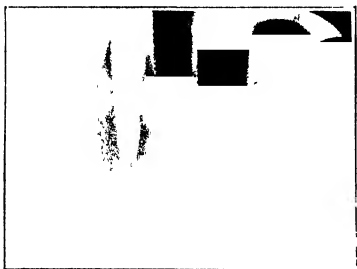


Fig. 37.



Fig. 38.



Fig. 39.



Fig. 40.



Fig. 41.

Figs. 37-41.—Illustrations of large alveolar abscesses.

or not they could be looked upon as indicative of infection. In forty-nine per cent the area was of such

size and appearance as to leave little doubt concerning the diagnosis of sepsis. It is interesting to compare these statistics with those reported by Black since his observations were made upon healthy individuals while those above mentioned were made upon medical cases. Since Black included in his number of shadowed teeth all those which showed evidence of bone absorption no matter how small, his forty-seven per cent found in relatively healthy individuals would compare perhaps with our eighty-one per cent found in individuals having systemic disorders. The actual percentage of devitalized teeth which become infected is, of course, even greater than these statistics would indicate, for the number lost by reason of infection are not included.



Fig. 42.

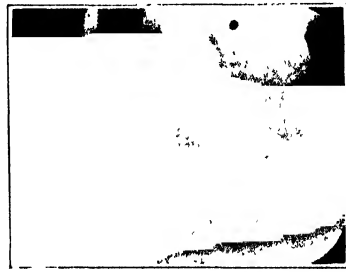


Fig. 43.

Figs. 42 and 43.—Osteomyelitis of the jaw derived from an infection at the roots of molar teeth which were extracted before roentgenogram was taken.

The tendency of devitalized teeth to abscess varies in different individuals. A few individuals with many devitalized teeth have no abscesses, while many individuals have an abscess at the root of every devitalized tooth. An individual with two or more devitalized is unusually fortunate if none are abscessed. Physicians in examining medical cases can feel relatively secure in assuming that teeth which have never been treated or filled, which are vital, and which appear outwardly normal are not the seat of infection. This is not invariably the case, however, for abscesses of pyorrheal origin are occasionally found at the roots of vital teeth. Occasionally in

the case of incisors irregularity of the teeth causes a thinning of the anterior wall of the alveolar process to such an extent that a very slight infection can reach the apex of the root. In this case a large abscess may be



Fig. 44.



Fig. 45.



Fig. 46.



Fig. 47.

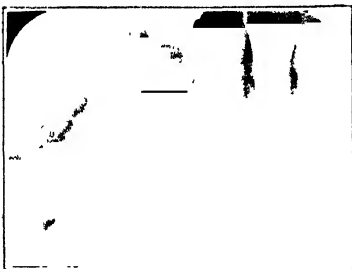


Fig. 48.



Fig. 49.

Figs. 44-49.—Illustration of the less active type of apical infection—the so-called granuloma. Note exostoses on roots shown in Fig. 49.

found at the root of a normal appearing vital tooth even when the gum shows surprisingly little evidence of disease. In a few cases observed by the writer a vital

nerve evidently passed through a granuloma. It is interesting to mention the fact that a vital nerve can be exposed to the infected material of an abscess for years



Fig. 50.



Fig. 51.



Fig. 52.



Fig. 53.

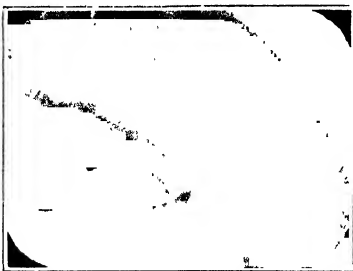


Fig. 54.



Fig. 55.

Figs. 50-55.—Typical example of oral sepsis in a patient with a great deal of dental work. Hardly a tooth can be found which is not the site of infection. Note the exostoses of the roots shown in Fig. 55 due to chronic irritation of the periodontal membrane.

and yet fail to cause a single symptom which attracts the attention of the patient. (See Fig. 17.)

Tooth pulp may be infected either directly as a result of the decay or treatment, or indirectly through the medium of the blood stream. The former source of infection is evidently common. That the latter source of infection is a real one is shown by the fact that abscesses are often found at the roots of teeth whose pulp chamber has not been perforated either by treatment or decay. For example, infection is occasionally found at the roots of unerupted teeth and at the roots of teeth whose pulp



Fig. 56.



Fig. 57.

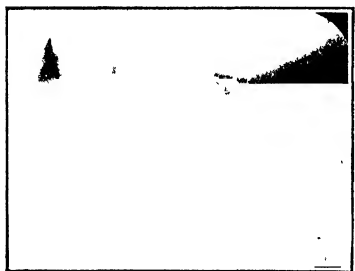


Fig. 58.



Fig. 59.

Figs. 56-59.—Example of the occurrence of numerous large abscesses in an individual with an excessive amount of dental work.

has been killed by trauma or by the proximity of large fillings. In rare instances abscesses are found at the roots of teeth which have been neither treated nor injured. In one individual that I observed three abscesses were found at the roots of the uninjured, untreated teeth.

The importance of filling the root canal to the very

tip of the apex after the devitalization of a tooth has been repeatedly emphasized by dentists. It is interesting in this connection to quote statistics recently re-



Fig. 60.



Fig. 61.



Fig. 62.

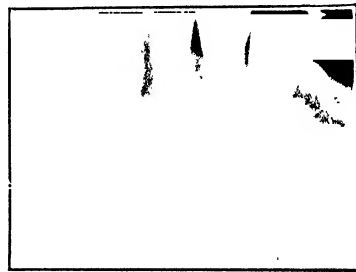


Fig. 63.



Fig. 64.

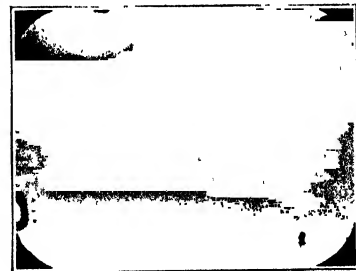


Fig. 65.

Figs. 60-65.—Example of an individual having a great deal of dental work, who shows relatively little periapical sepsis. This is a rather unusual finding and forms a marked contrast to the cases illustrated in Figs. 31 and 59.

ported by Black who found in examining healthy individuals that of 273 devitalized teeth in which the root canals were relatively well filled, only 23 were abscessed

(8 per cent); while of 580 teeth in which the root canal was poorly filled 379 were abscessed (65 per cent).

Medical cases which we have examined gave results which are somewhat different from the above. Of the



Fig. 66.



Fig. 67.



Fig. 68.



Fig. 69.

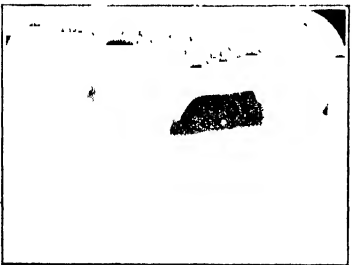


Fig. 70.

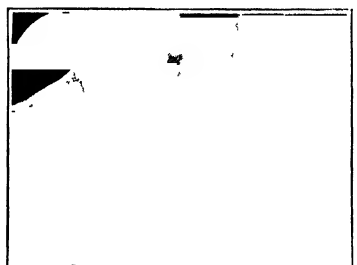


Fig. 71.

Figs. 66-71.—Illustration of the commonest source of periapical infection; namely, devitalized teeth with incompletely filled root canals.

devitalized teeth having poorly filled root canals 88 per cent were shadowed. In 32 per cent the shadows were extremely small. In 56 per cent the shadows were of

such size and appearance as to leave little doubt concerning the diagnosis of sepsis. Of the teeth having relatively well-filled root canals, 52 per cent were shadowed. In 32 per cent the shadows were extremely small. In 20 per cent the shadows were of such a size and appearance that they were thought to indicate the presence of sepsis almost positively. Teeth whose root canals had been reamed out with broaches and carefully filled to the very tip are not included in the above statistics for the reason

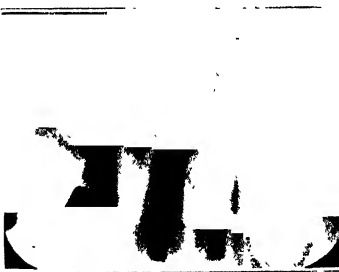


Fig. 72.

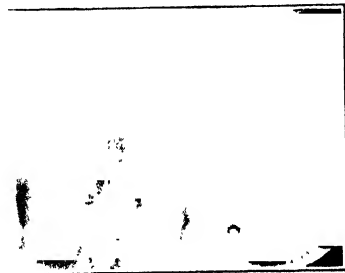


Fig. 73.



Fig. 74.

Figs. 72-74.—Illustration of abscesses due to perforation of root by pins.

that too few were found to give reliable percentages, and also because it was not possible in the majority of instances to determine whether such canals had been well filled immediately after devitalization of the nerve, or whether they were abscessed teeth which had been drained and filled a second time.

It is interesting to note that of all the crowned teeth examined in which an attempt had been made to leave

the root pulp vital 65 per cent were shadowed. In 42 per cent the shadows were very minute. In 23 per cent one could feel secure in making a positive diagnosis of sepsis. These findings are of interest in showing the necessity of care in the crowning of vital teeth. Among the cases that we have observed more sepsis was derived



Fig. 75.



Fig. 76.



Fig. 77.

Figs. 75-77. --Apical sepsis due to infection of the pulp by decay.

from teeth which were left apparently vital when crowned than from teeth which were purposely devitalized and left with the root canals only partly but relatively well filled.

It is also interesting to mention that 93 per cent of snags of teeth left by decay or after extraction were shadowed, and that in 67 per cent the shadows were relatively large in size.

The time at which root abscesses develop varies. One might presume that in the average case, it forms soon after the pulp has been destroyed. This, however, ap-

pears not always to be the case. Frequently, an acute abscess develops years after the devitalization of a tooth. In a very few instances in which teeth have been rayed a second time a granuloma has been found on the



Fig. 78.



Fig. 79.



Fig. 80.



Fig. 81.

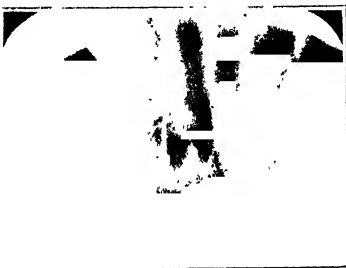


Fig. 82.

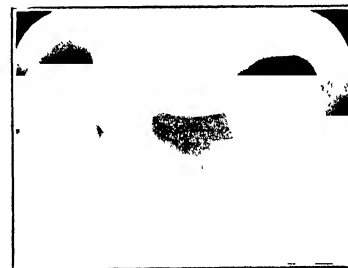


Fig. 83.

• Figs. 78-83.—Examples of root remnants which are the site of infection.

second examination which apparently did not exist when the first roentgenograms were made.

The mode of onset of apical infection varies. As a rule, it develops slowly and gradually, causing no symp-

toms by which its presence might be suspected. In rare instances it develops acutely soon after the filling of the root canal with the production of pus and a rapid destruction of bony tissue. This type of onset is usually

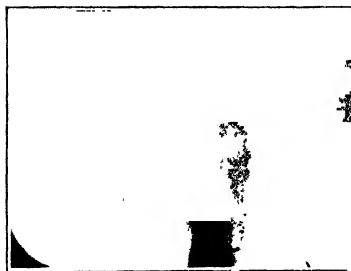


Fig. 84.

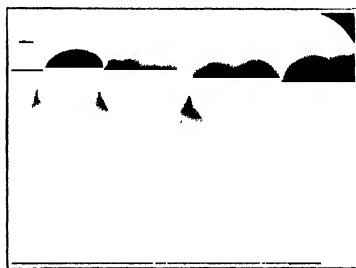


Fig. 85.

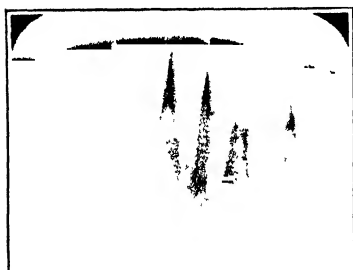


Fig. 86.



Fig. 87.

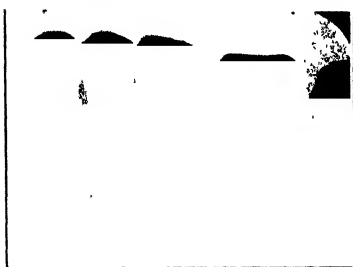


Fig. 88.

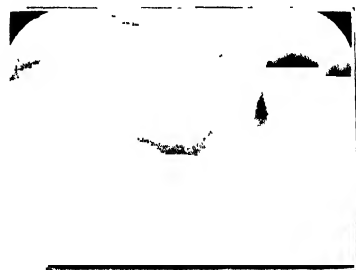


Fig. 89.

Figs. 84-89.—Examples of infection at the apices of filled teeth whose pulp was not intentionally destroyed.

associated with severe pain and the constitutional symptoms of an acute infection. Such an abscess may extend and involve the roots of the other teeth. It rarely be-

comes so extensive as to cause a definite osteomyelitis of the jaw. The rarity of this complication is probably to be accounted for by the fact that the distance of the abscess from the red marrow is relatively much greater than its distance from the surface of the bone, in fact there is very little red marrow in the jaw bones and this is separated from the alveolar process by a dense bony structure. In other words, an abscess usually breaks and discharges externally before it reaches the marrow. Abscesses occasionally reach the subperiosteal tissue, elevate the periosteum to a greater or less extent and break through and discharge externally for a time. Such a discharge may stop spontaneously and may recur from

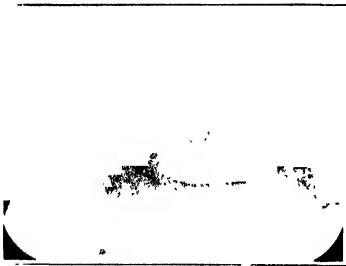


Fig. 90.



Fig. 91.

Figs. 90-91.—Examples of infection at the root apices of unerupted teeth.

time to time. Such an abscess is not inclined to heal permanently unless the tooth receives proper dental treatment. It may quiet down spontaneously, however, and develop into the so-called granuloma. This is more frequently the case with the smaller abscesses.

The acute development of alveolar sepsis as described above is the gross exception—not the rule. The usual mode of onset is insidious. In a vast majority of cases a small mass of pus or granulation tissue forms at the opening of the root canal. It varies in size from a minute mass to the size of a pea or larger and develops, as a rule, slowly and insidiously without causing pain or

discomfort and usually without even causing tenderness of the tooth. Such granulomata may exist unchanged for years, may vary in size from time to time due probably to trauma and changed states of health. They may heal and recur spontaneously and may develop into an acute abscess after years of quiescence.

The diagnosis of alveolar abscesses is made by the use of dental roentgenograms. Very little can be determined concerning their existence or extent except by this means: Unfortunately there is at present considerable divergence of opinion concerning the meaning of certain roentgen shadows. There are a few shadows which occur at or near the roots of the teeth not caused by in-

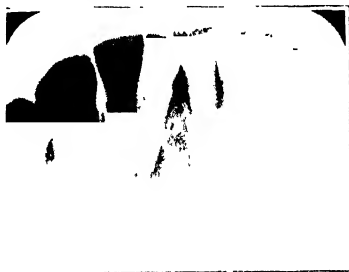


Fig. 92.

Fig. 92.—Example of periapical infection of teeth whose pulp was destroyed by severe trauma. Two of the root canals had been filled just previous to the taking of the roentgenograms.

fection which simulate those caused by infection. Such, however, are not common, and are not the usual source of contention. The chief source of divergent opinion concerns whether or not the shadows admittedly due to old abscesses or granulomata can be interpreted as proof of active infection. Many dentists believe that certain shadows are proof of past infection, but do not believe they can be looked upon as evidence of active infection. Some dentists take the extreme view that a shadow means sepsis only when there is pain, tenderness of the tooth, or other clinical manifestation to corroborate the diagnosis, and often disregard roentgen findings, which

as frequently happens, offer the only possible means by which root sepsis can be definitely proved. Unanimity of opinion upon the interpretation of dental roentgenograms is naturally of fundamental importance to both

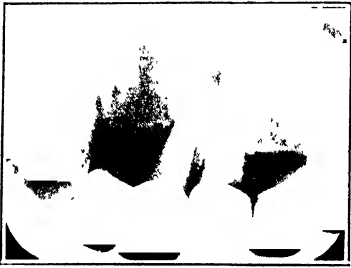


Fig. 93.



Fig. 94.



Fig. 95.



Fig. 96.



Fig. 97.

Figs. 93-97.—Examples of periapical infections of crowned teeth whose pulp was not purposely devitalized.

physicians and dentists, and the lack of this is a source of many contentions. It goes without saying that the final word concerning the interpretation of films should

be said by the dentist, but only by dentists who have made a careful study of the subject, who have had sufficient experience in this line to make their opinion reliable, and who look upon sepsis as a serious condition. .

There is considerable evidence, although at present incomplete, to show that shadows at the apices of teeth caused by the replacement of bone by soft tissue almost invariably indicate the presence of infection. Primarily, it may be said that a growth of organisms has been obtained in culture by a number of observers from such localities, and bacteria have been demonstrated microscopically in stained smears of the material scraped from the roots of such teeth. These studies, however,

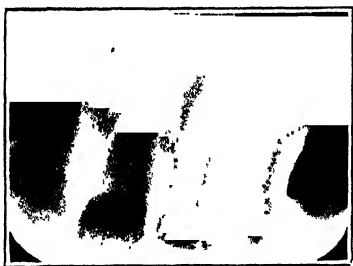


Fig. 98.

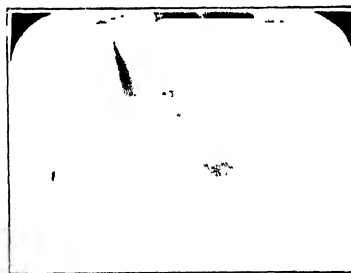


Fig. 99.

Figs. 98-99.—Examples of periapical infection of untreated teeth. Such teeth usually give a history of injury, but this is not always the case.

are not as yet complete enough to admit conclusions which might be looked upon as final.

We wish to suggest a second line of evidence which, though indirect, seems rather convincing. It is a fact well known to pathologists that granulating lesions in bone almost invariably heal and become organized into solid bone unless there is a definite reason for their not doing so. The cause of failure of such lesions to ossify is usually infection or a foreign body or a sequestrum. If such obstacles to healing are removed, organization and ossification of the infected areas usually take place rapidly.

The newly formed bone differs in its histologic struc-

ture and in gross architecture from normal bone. It differs usually however, in being abnormally dense, so that instead of casting roentgen shadows showing areas of diminished density, it more often casts shadows indicating areas of increased density. Tubercles of bone become organized and ossified as soon as the active tuberculous infection is overcome. Gunmata of bone are usually organized and transformed into solid dense bone after antispecific treatment has been properly instituted. Likewise the granulation tissue formed in os-



Fig. 100.



Fig. 101.

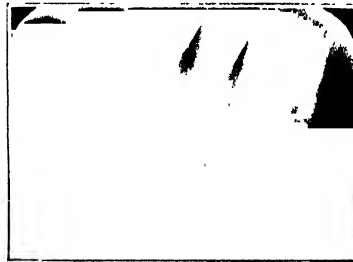


Fig. 102.

Figs. 100-102.- Illustrations of teeth showing absorption of the tips of the root apices (Figs. 100 and 101) and necrosis of the apex (Fig. 102).

teomyelitis is often so completely ossified after the sequestrum is removed and infection eliminated by drainage, etc., that x-ray pictures often show relatively little difference between it and the corresponding bone of the opposite side. These gross examples of the healing of bone lesions lead one to infer that areas relatively so minute as granulomata at the roots of the teeth should

ossify almost invariably and rapidly unless there exists a very definite cause for their not so doing. The cause in this case would appear usually to be infection. In harmony with this view is the well-recognized fact that drainage of a root abscess followed by the complete filling of the root canal often leads to disappearance of the rarefaction shadow caused by it. The organization is



Fig. 103.



Fig. 104.

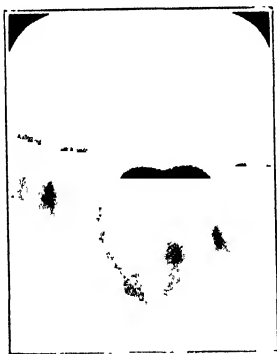


Fig. 105.

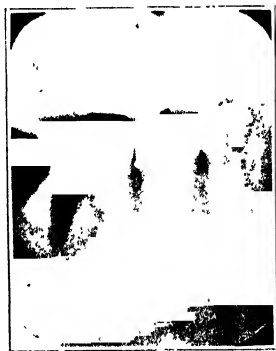


Fig. 106.

Figs. 103-106.—Illustrations of several teeth showing one or more completely necrotic roots.

often so complete that roentgenograms may show relatively little evidence of its past existence. It is, of course, not always possible to obtain this result. If the periosteum of the root has been extensively destroyed so that the apex of the root or the entire root is necrotic, the condition remaining after drainage or after the use of antiseptics is analogous in many respects to osteo-



Fig. 107.

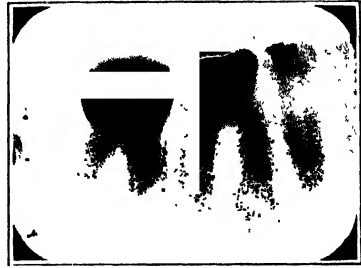


Fig. 108.



Fig. 109.

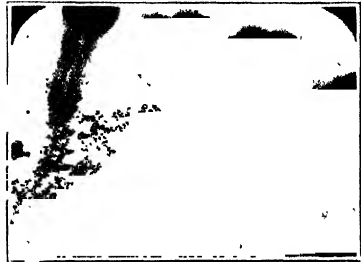


Fig. 110.



Fig. 111.



Fig. 112.



Fig. 113.

Figs. 107-113.—Illustrations of shadows indicating areas of increased density in the alveolar process as a result of healing of infected areas. Shadows of such density as the above are not commonly observed.

myelitis in which healing is prevented by a sequestrum.

Frequently it appears to one that active infection can not exist for an indefinite period of time in granulation tissue or in the pus of a chronic abscess such as an alveolar abscess unless it be repeatedly reinfected from some source. The organisms in granulation tissue or in chronic abscesses are exposed to the defensive action of the blood, lymphocytes, leucocytes, connective-tissue

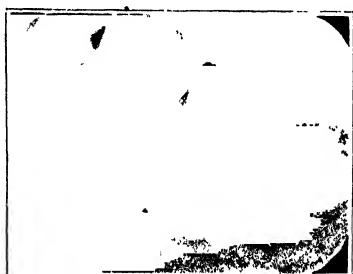


Fig. 114.



Fig. 115.

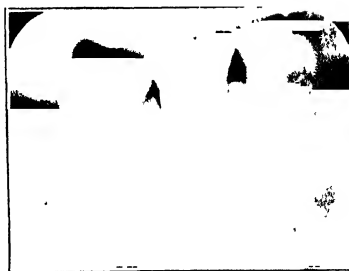


Fig. 116.

Figs. 114-116.—Illustrations of radiopaque areas which simulate the healed abscesses shown in Figs. 107-113.

cells, etc., and in the course of time are perhaps destroyed by them. Organisms in the root canal of non-vital teeth or in the little cavities in necrotic portions of the root, however, are often safely removed from the defensive mechanism of the body and favorably located to repeatedly infect a granulating lesion around it. It is probably for this reason that the complete filling of a

root canal or the removal of the necrotic tip of a root often is followed by organization of an abscess.

. The above fact has been made use of for several years by dentists in their efforts to clear up root sepsis by



Fig. 117.

Fig. 117. Illustration of roentgenogram showing bone of increased density due apparently to increased stress transmitted by the tooth during mastication. Areas of increased density, such as these, are not uncommonly observed.

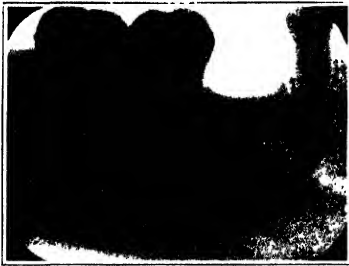


Fig. 118.



Fig. 119.

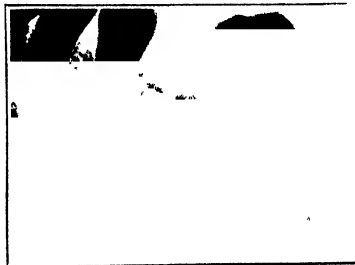


Fig. 120.

Figs. 118-120.—Examples of root remnants which have apparently become encysted.

conservative means. The question as to whether or not this method of treatment is thorough and reliable, how-

ever, is still an open one. Several observers have been able to obtain cultures of streptococci from the roots of teeth treated in this way even after nearly perfect organization of the granuloma could be demonstrated roentgenologically. On the basis of this work such teeth would appear theoretically, therefore, to remain possible sources of systemic infection. Numerous patients under our observation with systemic disturbances, however, have been very definitely improved in general health after conservative methods of treatment.

The question as to the best method of treatment might yet be considered an open one and for the present at least the choice of method in each individual case might be based in part upon the apparent possibility of eradicating sepsis by conservative means and in part upon the gravity of the systemic ill for which the teeth might appear to have been contributing causes. The following illustrations (Figs. 121 to 151) show the value of roentgenograms in determining the end result of conservative methods of treatment.

FIGS. 121-151.

Examples showing the effect of palliative measures in the treatment of alveolar abscesses. Note the variability in the final result. In many instances the abscesses were organized to a greater or lesser degree after drainage followed by the use of antiseptics and complete filling of the root canal; in others no marked evidence of organization could be observed in roentgenograms. These prints are not perfect reproductions of the roentgenograms, and make the palliative measures appear more favorable than is actually portrayed in the films. The systemic condition of the patient in several instances improved markedly after the palliative measures illustrated here.

We do not wish to comment upon the adequacy of the results shown here since this is as yet an open question and may not prove so favorable as the roentgenograms might lead one to believe.

The root canal work shown in these illustrations was done by Drs. J. E. Huff, Jerome Stuart, H. V. Brockett, A. C. Erdman, M. C. Carpenter, R. N. Seibel, F. W. Franklin, H. S. Lowry, E. M. Hall, D. R. Taylor, and C. J. Davis.

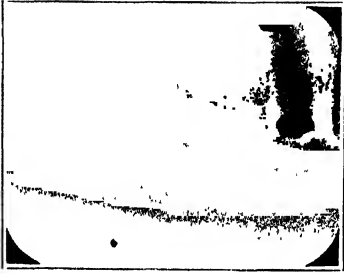


Fig. 121.

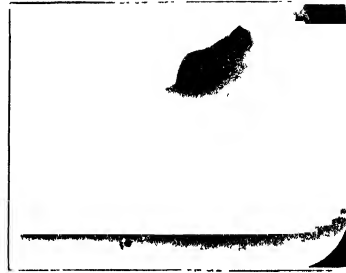


Fig. 122.



Fig. 123.



Fig. 124

Fig. 121.—Roentgenogram taken immediately after drainage and filling of the root canal.

Fig. 122.—Roentgenogram of the same tooth approximately one year later.

Fig. 123.—Roentgenogram taken just before drainage and filling of the root canal.

Fig. 124.—Roentgenogram of the same tooth two months later.



Fig. 125.

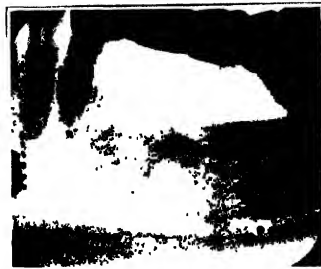


Fig. 126.



Fig. 127.



Fig. 128.

Fig. 125.—Roentgenogram showing small area of apical sepsis.

Fig. 126.—Roentgenogram of same tooth taken approximately six months after its treatment.

Fig. 127.—Roentgenogram showing small area of apical sepsis.

Fig. 128.—Roentgenogram showing same tooth approximately three months after its treatment.



Fig. 129.



Fig. 130.



Fig. 131.

Fig. 129.—Roentgenogram showing apical infection.

Fig. 130.—Roentgenogram taken shortly after the filling of the root canal.

Fig. 131.—Roentgenogram taken approximately eight months later.

This abscess was organized in large degree in spite of the fact that the root canal was not perfectly filled and the tip of the apex was very evidently necrotic.



Fig. 132.



Fig. 133.



Fig. 134.

Fig. 132.—Roentgenogram showing an abscess at the root of a crowned left upper incisor. The abscess had burrowed to the right and discharged near the root apex of the right upper incisor. Bismuth had been injected into the sinus some months previous and shows as a dense opacity in the roentgenogram.

Fig. 133.—Roentgenogram taken three months after irrigation through the root canal of the left incisor followed by filling of the root canal.

Fig. 134.—Roentgenogram taken six months after treatment.

Organization of the abscesses in this case was very rapid. A change considerable in degree was noticed after a few weeks.



Fig. 135.



Fig. 136.



Fig. 137.



Fig. 138.



Fig. 139.

Fig. 135.—Roentgenogram showing apical infection at the root of a lower molar tooth.

Fig. 136.—Roentgenogram taken approximately six months after drainage and filling of the root canal.

Fig. 137.—Roentgenogram showing sepsis at the apices of two lower molar teeth.

Fig. 138.—Roentgenogram taken six months later.

Fig. 139.—Roentgenogram taken approximately one year after completion of treatment.



Fig. 140.



Fig. 141.



Fig. 142.



Fig. 143.

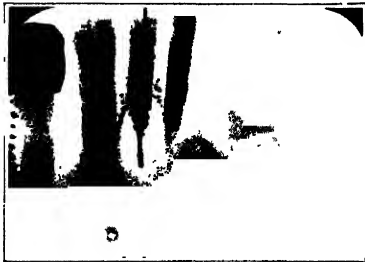


Fig. 144.



Fig. 145.



Fig. 146.

Fig. 140.—Roentgenogram showing large alveolar abscess.

Fig. 141.—Roentgenogram taken ten weeks after treatment.

Fig. 142.—Roentgenogram taken four months after complete filling of root canal.

Fig. 143.—Roentgenogram showing an abscess at the roots of two lower incisor teeth.

Fig. 144.—Roentgenogram taken during treatment of abscess.

Fig. 145.—Roentgenogram taken immediately after completion of treatment.

Fig. 146.—Roentgenogram taken approximately one year after completion of treatment. Failure in result due apparently to fact that tooth was necrotic.

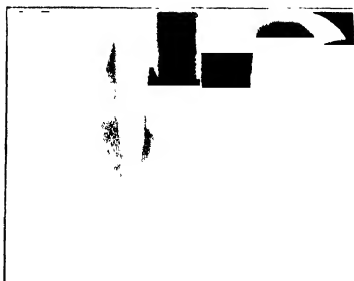


Fig. 147.



Fig. 148.

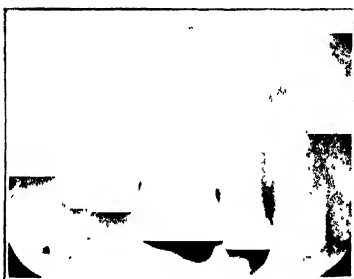


Fig. 149.



Fig. 150.

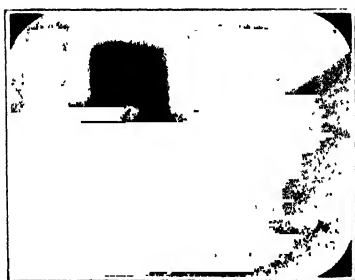


Fig. 151.



Fig. 152.

Fig. 147.—Roentgenogram showing large discharging alveolar abscess at the apex of a lower incisor tooth.

Fig. 148.—Roentgenogram taken approximately four months after completion of treatment. No evidence of healing is discernible in spite of the fact that the abscess was irrigated and drained and the root canal filled to the apex.

Fig. 149.—Roentgenogram showing an abscess at the root of an upper bicuspoid tooth.

Fig. 150.—Roentgenogram of the same tooth taken approximately six months after treatment. No organization. Root canal not properly filled.

Fig. 151.—Roentgenogram showing an abscessed lower molar tooth.

Fig. 152.—Roentgenogram taken approximately three months after completion of treatment.

FIGS. 153-161.

Examples of careful root canal work. More is accomplished by careful laborious work such as the following if it is directed towards prevention of sepsis rather than towards its cure. It is stated by dentists that periapical sepsis can usually be prevented if root canals are filled in this way immediately after devitalization of the pulp.

The best interest of the patient demands that root canals be filled in this way and that the thoroughness of the result be determined by the use of roentgenograms.

Note multiple apical foramina shown in Fig. 156 and lateral accessory foramina shown in Figs. 157 and 158.

Roentgenograms taken by Dr. J. A. Sawhill.



Fig. 153.



Fig. 154.



Fig. 155.



Fig. 156.



Fig. 157.



Fig. 158.

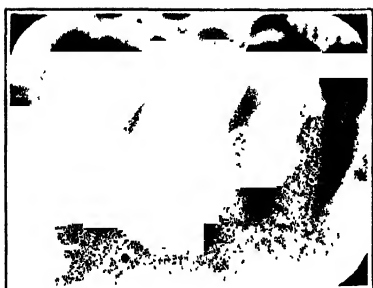


Fig. 159.



Fig. 160.



Fig. 161.

CHAPTER IV

METASTATIC INFECTION

The spread of infection from a chronic focus occurs in three ways, by direct extension to adjacent tissues, by transportation along mucous or serous surfaces, and by metastatic infection through the medium of the blood stream and lymph with the final involvement of distant organs. The last mentioned type of infection is by far the most important.

The animal organism gains an immunity against chronic infections and is often able under the usual conditions of life to keep them localized. Invasion of the blood and spread from chronic foci occur usually during periods when resistance is lowered by the effect of such conditions as fatigue, exposure, digestive disturbance, lack of proper nourishment, overindulgence in alcohol, pregnancy and lactation, diabetes, anemia, debilitating diseases, acute and chronic infections, etc. The important influences of these factors upon the defensive mechanism has been repeatedly observed both clinically and experimentally. One might quote in this connection the discovery of Pasteur that fowls which are naturally immune to anthrax become susceptible to the disease if the body temperature is reduced by a cold bath, also the discovery of Charrin and Roger that white rats which enjoy a similar immunity against anthrax become quite susceptible if they are exhausted by physical exertion. Organisms harbored in such chronic foci of infection as alveolar abscesses are held local a large part of the time. They repeatedly find the defenses of the body below par, however, and find abundant opportunity

to multiply rapidly, invade the blood and gain lodgement in distant structures.

A variety of organisms can be obtained in culture from periodontal infections. *Streptococcus viridans* is found more constantly in the deeper pyorrhea pockets and in alveolar abscesses than other organisms, and usually predominates in number. Of the metastatic infections attributable to focal infection, those caused by the streptococcus group appear to be the most important and have been the subject of most extended study.

A new and interesting conception of infection and of diseases caused by streptococci has been suggested by Rosenow, Billings, and their coworkers as a result of extensive laboratory and clinical investigations carried on during the past few years. The conclusions which these observers have drawn have not been accepted in their entirety, and have recently been the subject of much discussion and controversy. While some of the views which they express may prove fallacious, others which are of fundamental importance will undoubtedly stand. The more important suggestions which they have made are: First, that the number of diseases caused by streptococci is greater than was formerly supposed; second, that streptococci may acquire affinities for certain tissues, which in many instances is remarkably specific; third, that streptococci may change in their selective affinities, morphology and cultural characteristics and biologic reactions after cultivation in artificial media or after growth in animal tissues.

The fact that streptococci can acquire an affinity for a certain specific tissue was suggested earlier by Forssner who working with a strain of streptococcus obtained from an abscess which had no particular affinity for kidney tissue stated that he was able to develop such an affinity in the organism by taking cultures from the kidney lesions of animals which he had inoculated with the

nonspecific culture. This affinity was lost after several transplants in bouillon.

Billings and Rosenow conclude from their observations that members of the streptococcus pneumococcus group may develop affinities for certain tissues, not only while growing in those particular tissues, but also while growing in primary foci of infection. They state that the organisms obtained from the infected tissue of patients with such diseases as appendicitis, cholecystitis, gastric and duodenal ulcer, glomerular nephritis, rheumatism, endocarditis, etc., show striking similarities in their elective affinities to the organisms obtained from the related foci of infection. They found the elective affinity less marked, however, in the strains isolated from the primary foci.

The interesting deduction is made by Rosenow that since bacteria which have grown in given tissues acquire an affinity for that tissue, the likelihood of such organisms to involve structures of a different nature is relatively slight and that a focus in a specialized tissue, therefore, would seem less important as a distributor of bacteria than the primary focus. He believes that bacteria growing in primary foci not localized in specialized tissue can change in their affinities and invade one type of tissue after another. For example, bacteria which have localized in joints and acquired a special elective affinity for joint structures are less likely to spread to other organs such as the appendix, kidney, gastric mucosa, etc., than organisms localized in nonspecialized tissues, such as tonsillar crypts, alveolar abscesses, etc., where they are constantly subjected to varying conditions of oxygen tension, and where they may be changed thereby in their nature and affinities just as they may be changed by various artificial conditions when grown in culture media.

The above conclusions concerning selective affinity of

organisms and transmutation of strains are not altogether radically opposed to views held formerly. It has long been known that the tendency of organisms to localize depends to a certain extent on virulence, and that the virulence of an organism is changed by environment. Rosenow's elaboration has been so extensive, however, as to almost revolutionize former views concerning infection. It is quite proper that conclusions so interesting and of such fundamental importance be carefully analyzed by other observers, and that their full acceptance await repeated verification.

While Rosenow's interesting experiments suggest in detail the mechanism by which certain tissues are infected, their acceptance is not essential to the popular view that many acute and chronic diseases are attributable to infection distributed by small foci of infection and that streptococci distributed by them may cause systemic diseases whose gravity seems out of all proportion to the size and activity of the original apparently trivial focus.

There is also little question concerning the fact that streptococci which have been isolated from tissues in a great variety of diseases upon inoculation into rabbits produce in a majority of cases pathologic changes similar to those in the tissues from which the organisms were originally obtained; nor concerning the fact that the predominating organism isolated from a focus of infection may produce upon inoculation into animals the same pathologic changes as the organism obtained from the diseased tissue which derived its infection from the focus.

The following diseases, often streptococcic in origin, may originate as a metastatic infection from chronic foci of infection such as pyorrhea alveolaris and alveolar abscesses: Rheumatic fever, acute and chronic infectious arthritis, myositis, bursitis, neuritis, iritis, and other inflam-

matory diseases of the eye including possibly neuroretinitis, vegetative endocarditis, ulcerative endocarditis, myocarditis, pericarditis, phlebitis, peritonitis, chorea, spinal myelitis, meningitis, acute and chronic nephritis, acute and chronic appendicitis, cholecystitis, gastric and duodenal ulcer, pancreatitis, thyroiditis, erythema nodosum, herpes zoster, osteomyelitis, periostitis, pneumonia, pleurisy, empyema, septicemia, erysipelas, cellulitis, lymphadenitis, etc.

It is generally recognized and has been emphasized by Rosenow that streptococci have general virulence as well as selective affinities for certain tissues. Streptococci obtained from acute arthritis upon injection in rabbits infect not only the joints, but also, in a minority of cases, other organs, such as gall bladder, gastric mucosa, endocardium, myocardium, etc. The general virulence of an organism is of considerable importance in the pathogenesis of disease and often causes spread of an infection to tissues for which it has no selective affinity. This is especially the case during periods when resistance is low.

Resistance may be lowered locally as well as generally. It may be lowered locally by many types of trauma, strain, intoxication, lack of local nourishment, local interference with blood supply, etc. To illustrate this factor it may be mentioned that individuals with rheumatism frequently notice the involvement of a new joint following some slight injury to the joint. In fact, in chronic arthritis the joints most commonly involved are those subjected to frequent slight trauma. The joints of the fingers are commonly involved in individuals who use their hands a great deal, the hip joints in delivery men who frequently jump from their wagons, the ankle joints in individuals who have flatfoot with the unusual strain on the ligaments caused by this condition, the knees in individuals whose knee joints are strained by

reason of genu valgus, the joints of the spine in individuals with postural defects, etc. These examples illustrate the influence of strain and trauma in determining the localization of organisms which already have affinities for given types of tissue (in the examples mentioned affinity for joint structure). Local injury may also lead to the infection of tissues by organisms which have no especial predilection for them. For example, local injury to a bone may cause osteomyelitis by rendering the tis-



Fig. 162.

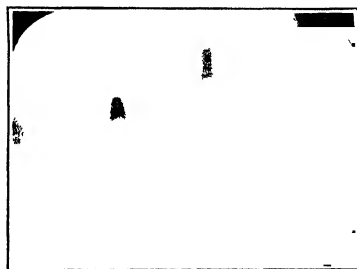


Fig. 163.



Fig. 164.

Fig. 162.—Case of chronic migratory polyarthritis of several months' duration which was completely relieved by the extraction of the tooth shown in the above illustration.

Fig. 163.—Case of multiple neuritis of some six months' duration which was completely relieved by the extraction of the tooth shown in above illustration.

Fig. 164.—Case of recurrent pain in gall bladder region associated with occasional slight jaundice which was apparently relieved by the extraction of the tooth shown in above illustration. Cholecystotomy had been advised and refused by the patient before the tooth was extracted.

sues suitable for the growth of organisms which originally had no apparent specific affinity for bone marrow. Opie was able to combine chloroform poisoning, which causes a high grade of liver necrosis, with the inocula-

tion of living organisms of varying virulence and produce pathologic changes in the liver analogous to acute yellow atrophy and cirrhosis of the liver. The changes observed were apparently dependent in part upon the degree of injury to the liver cells produced by the chloroform, and in part upon the general virulence of the organisms used. He was not able to produce changes typical of either of the above diseases by chloroform poisoning alone or by infection alone. His deduction that the above diseases in humans might be due to a combined effect of toxemia and infection appears to be well founded. The strain of organisms used, of course, had no specific affinity for liver tissue. Opie's experiments explain possibly the fact that alcohol, which figures so frequently in the etiology of hepatic cirrhosis, may be indulged in excessively over a period of years by many individuals without the appearance of cirrhotic change. Possibly the liver of a normal individual can tolerate an amount of abuse by alcohol and other poisons which might lead to chronic inflammatory changes ending in liver cirrhosis in individuals suffering from chronic infection such as oral sepsis, tuberculosis, syphilis, etc.

The various organs and tissues of the body are, of course, repeatedly subjected to conditions which increase their susceptibility to infection, such as local strain, exhaustion, trauma, local interference with the blood supply, obstruction of the excretory ducts by stones, and by the effect of toxins and poisons of many kinds. Such conditions might be tolerated by normal individuals but might lead to local infection and serious disease in individuals having chronic infections. It is perhaps possible in this way to account for the existence of widespread systemic disease in some individuals having focal infections and its total absence in others who have similar foci. Chronic infections are probably of more serious moment in individuals whose systems are subject to injury

by other agents. Conversely, abuses of any sort would appear of more serious moment in individuals having chronic infections than in individuals who are free from them.

The importance of oral sepsis as a source of metastatic infection is not small. It is the most common of the chronic infections, therefore one of the more important. It is a less apparent source of disease than the tonsils. The difference is, however, probably more apparent than real.

Systemic infection attributable to oral sepsis is usually chronic and insidious in onset. The effects are so insidious that the resulting pathologic changes are often extreme in grade before they are noticed clinically. For this reason the treatment of oral sepsis does not always give the relief that might be hoped for.

The inflammatory process in a given lesion, arthritis, for example, often continues long after the primary focus of infection is removed. This may be due in part to the fact that the organisms have produced local pathologic changes in the tissues which are favorable to their growth and in part to the fact suggested by Welch that organisms in their struggle for existence in the tissues of an individual may acquire the faculty of protecting themselves against his defensive mechanism. Either factor might render difficult or impossible their thorough and complete eradication from the tissues.

CHAPTER V

NONRELATED INFECTION AS INFLUENCED BY ORAL SEPSIS

It is generally recognized that when an individual has two or more diseases, one may influence the other occasionally to the apparent advantage of the patient, but more frequently to his disadvantage. In other words, increased susceptibility to one organism may be caused by infection with another. This can be well illustrated by familiar clinical examples. Latent tuberculosis may become active after an attack of tonsillitis, bronchitis, or la grippe. It was mentioned in a previous chapter that a patient who had latent specific disease was covered with a copper-colored rash and gave a positive Wassermann reaction at the end of the first week of convalescence from typhoid fever. Urethritis is often unfavorably influenced by the appearance of a new disease, and apparently cured cases may recur after an attack of tonsillitis or la grippe. In a patient observed by us an attack of bronchopneumonia followed several days after the development of an acute alveolar abscess which required lancing. Several weeks afterward the patient had a recurrence of active tuberculosis which had been latent for many years.

It is frequently observed that infections of a milder nature, such as chronic oral sepsis, chronic tonsillitis, etc., may have an untoward influence upon the course of other unrelated infectious diseases with which an individual may be afflicted. This influence is often quite marked. The ill effect of chronic mild infections of the nose and throat upon tuberculous individuals is recognized, and proper local measures are looked upon as an

important part in the regime for the treatment of tuberculosis. Oral sepsis also may exert an unfavorable influence upon the course of tuberculosis and marked improvement may follow the removal of abscessed teeth. According to the observations of my friend, Dr. Richard L. Sutton, chronic infections of the skin, such as acne and furunculosis, are often favorably influenced by the removal of focal infections in which streptococci may be the dominant organisms. In a patient with staphylococcus aureus septicemia a fall of four degrees in temperature occurred several hours after the removal of a chronically abscessed tooth which showed a pure growth of streptococcus viridans on culture. The temperature

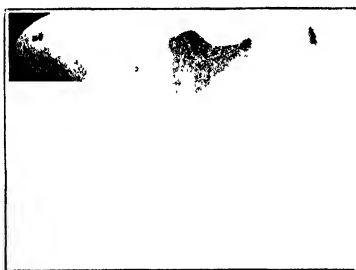


Fig. 165.

Fig. 165.—Case of staphylococcus septicemia of about two months' duration. Temperature, which has been constantly reaching 102° or over each day, dropped to normal a few hours after the extraction of the tooth shown in the above illustration and remained normal for ten days. The case ended in complete recovery. The root apices showed streptococcus viridans in pure growth when cultured.

remained normal for ten days following this, and the case eventually recovered. The temperature had reached 102° each day for the previous two months. (Fig. 165.)

The ill effect of focal infections upon individuals with syphilis of the nervous system is very great indeed. This will be dealt with in some detail on account of its great practical importance. The Wassermann test made as a routine in one thousand medical cases observed in office practice was positive in 11 per cent. Involvement of the nervous system was found clinically and verified by lumbar puncture in over 50 per cent of these; that is, in ap-

proximately 6 per cent of the total number of medical cases examined. The overwhelming majority of these had several alveolar abscesses. The occurrence of syphilis of the central nervous system and alveolar abscesses in the same individual is, therefore, a very common occurrence. The fact that nonrelated infections may rapidly hasten the course and augment the symptoms of tabes dorsalis is well illustrated by the following case history which may be reported briefly as follows:

Patient, male, age thirty-five, had been treated for two years for tabes dorsalis. Before treatment was started he had noticed slight ataxia and bladder disturbance and was subject to mild attacks of darting pain in the legs. He improved very satisfactorily under antispecific treatment, and after he had been free from pain and bladder disturbance for more than a year, he had an attack of acute tonsillitis. With the onset of this there followed a recurrence of the most severe lightning pains which the patient had ever experienced. Morphine and salicylates to the limit were required to keep the patient out of agony. There was nothing to account for the sudden recurrence of acute symptoms unless it were tonsillitis. A tonsillar abscess developed, and when this was lanced, lightning pains ceased immediately and almost completely. A few slight pains were noticed until the tonsils were removed some three weeks later. During two years, since that time, the patient has been practically free from any active symptoms of tabes and has required very little treatment of any kind to prevent recurrence of his previous symptoms.

The above case was very striking indeed and convincing of the fact that unrelated infections may have an untoward influence on the course of tabes dorsalis which is by no means small. All the symptoms in early cases, including lightning pains, numbness, paresthesia, ataxia, visceral disturbances, etc., appear to be made worse by them.

For two years all cases of syphilis that we have treated have been urged to have every source of infection radically removed if possible. The patients with tabes have been benefited by this almost without exception. Some who were running rather unfavorable courses, in spite of antisyphilitic treatment, began to improve immediately. The following is mentioned as a typical example:

Patient, age fifty years; case had been diagnosed general paresis and had been given thorough antisyphilitic treatment for two years. When he came under our observation, he was unable to attend to business, and had to spend most of his afternoons in bed. All known methods of treatment had been pushed to the limit and it appeared that very little improvement could be expected from further antisyphilitic therapy. The patient had large tonsils and several abscessed teeth. These were removed. The patient improved steadily and to an unbelievable degree from the date of the above operations. Within less than a month he was able to engage in active business again. He has continued mild antisyphilitic treatment, and has remained in active business since that time.

If thorough and radical removal of all sources of chronic infection precedes the antisyphilitic treatment of tabes and paresis the improvement is often so striking and gratifying to the patient that it is occasionally difficult to persuade him to undergo the hardships of intensive treatment with salvarsan and mercury. The following case is mentioned as a typical result of removal of focal infection in a previously untreated patient with tabes dorsalis whose disease had been running a rapidly progressive course.

Patient, male, age forty-five, was brought in with a diagnosis of tabes dorsalis. Physical and laboratory examinations left no doubt concerning this. The onset had been stormy and for three weeks the patient had been

fairly convulsed with lightning pains and had hardly been able to control himself. He was so nervous and irritable at the time of the examination that it was almost impossible to get him to remain quiet long enough to obtain a specimen of blood for examination. Lumbar puncture could not have been done without general anesthesia. Dental films showed an advanced degree of sepsis. The abscessed teeth were all extracted. Within a few days the patient was so improved that he went back to work. He felt and acted like a well man even though antispecific treatment had not yet been administered.

Many cases similar to the above could be cited. These are not picked cases, but represent only an average experience. Nearly all the cases of *tabes dorsalis* that we have treated have been early cases, and those in whom it has been possible to remove every other source of infection have responded splendidly to antispecific treatment, and have apparently required less treatment than was formerly used to stop the progress of the disease. Rapidly progressive cases of *tabes* and *paresis* usually show many sources of infection and usually run a milder course which can be controlled by antispecific treatment if all such infections are radically removed. Severe lightning pains occur usually in those patients who have many foci of infection, and they can often be reduced or remarkably relieved by removal of all such foci.

Chronic sepsis probably takes no part in the pathogenesis of *tabes dorsalis* except as that of an unrelated infection which favors the advance of the specific process. All of the patients on whom observations were made gave positive Wassermann tests before and after the removal of focal infections, and the changes observed in the spinal fluid did not differ strikingly from those which might have been expected to follow the antisymphilitic measures which were employed.

Removal of focal infections as part of the regime in the treatment of syphilis is also of value in facilitating the use of mercury and potassium iodide and in rendering their use less harmful. Mercury and potassium iodide in therapy exert an unfavorable influence upon infections in the alveolar process and in the throat and apparently also on pyogenic infection in other localities. In many instances they cause trivial infections to develop rapidly and give rise to definite toxemias. It is impossible to push mercury and potassium iodide to the physiologic limit in the face of severe oral sepsis or badly infected tonsils without increasing the local trouble and rendering the systemic effect of the infections more serious. Many of the untoward effects of mercury and potassium iodide are without question due to their action in stirring up the latent infection. For this reason the radical treatment of oral sepsis with the aid of roentgenograms, the removal of infected tonsils, and all other infections is strongly indicated as an adjunct to the treatment of syphilis. When this is done, mercury and potassium iodide pushed to the physiologic limit appear to be relatively harmless.

CHAPTER VI

TOXIC EFFECT OF ORAL SEPSIS

A true toxin is not formed by any of the organisms which ordinarily take part in the pathology of oral sepsis. Killed cultures of the organisms, or extracts of the organisms, or the culture media in which they have grown may be injected in large quantities into healthy guinea pigs without immediate gross ill effect. These organisms differ strikingly in this respect from tetanus and diphtheria bacilli, each of which produces a true toxin which is poisonous in high degree even when administered subcutaneously to animals in very minute doses. The products formed by certain organisms which inhabit the mouth may be extremely toxic, however, through an entirely different mechanism; namely, through the development in an individual of a condition known as allergy. This term, introduced by von Pirquet, is used to designate the changed condition of humans or animals caused by infectious diseases or produced by inoculation with alien proteins which causes the individual to react in a peculiar way if the bacteria responsible for the infectious disease or if the protein with which he may have been inoculated is reintroduced into the circulation. The ensuing reaction is in some respects protective and beneficial, but in other respects it may be harmful and even dangerous.

• Allergy and its effects are well illustrated by the action of tuberculin. Killed tubercle bacilli or their extracts, or media in which tubercle bacilli have grown may be injected in large amounts into normal untreated animals without gross immediate ill effect. If even minute quantities be injected into animals or individuals with

tuberculosis, however, the effect is entirely different. Instead of its being apparently inert, it causes a reaction which may result quickly in death. The changed condition of the animal produced by the tuberculous infection which renders it sensitive to the products of the tubercle bacillus is the condition known as allergy. Several striking phenomena follow the introduction of tuberculin into tuberculous individuals or animals which never appear in the nontuberculous. These are: first, pain, swelling, redness, and sometimes necrosis, at the site of the inoculation; second, a rise of temperature and pulse rate and general symptoms of toxemia; third, inflammatory changes at the site of all active tuberculous lesions. This last phase of reaction may be slight or may be well marked. It may hasten the breaking down of tuberculous tissue and lead to rapid spread of the disease.

Some individuals with tuberculosis become so sensitive to products of the tubercle bacillus that amounts of tuberculin as minute as one one-thousandth of a milligram are sufficient to produce severe reactions. Others tolerate larger amounts.

A reaction as above described can be produced in tuberculous animals by the subcutaneous injection of living tubercle bacilli as well as by extracts of the organisms. If tuberculous guinea pigs are inoculated with living tubercle bacilli, a local and general reaction results similar in its essentials to the reaction produced by extracts of dead bacilli. If a localized tuberculous lesion in humans (such as a tuberculous joint) is subjected to massage and manipulation, living tubercle bacilli and their products gain entry into the circulation and a transitory rise of temperature and other symptoms of reaction follow.

The above well-known facts regarding tuberculous infection and the sensitization produced by it are true of infections in general. An attack of typhoid, for example,

renders an individual sensitive to the protein of the typhoid bacillus, an attack of furunculosis renders one sensitive to the staphylococcus aureus, etc., so that individuals having these diseases usually show a local, general, and focal reaction if they are inoculated with killed cultures of the offending bacteria. This may temporarily increase the inflammatory processes, the severity of which would appear to depend to a certain extent upon the quantity of bacteria injected.

Individuals with chronic furunculosis react and show temporary exacerbation of the disease, not only when inoculated with an excessive quantity of killed staphylococci, but also when they are subjected to the effect of an increased number of living bacteria due to the development of a fresh furuncle. This often causes both a general and a focal reaction. For example, with the development of a fresh furuncle there may appear fever, malaise, etc. (general reaction) and an exacerbation in the inflammatory process in the healing furuncles (focal reaction), in fact, signs of activity, such as itching, redness, etc., may appear in the scars of recently healed furuncles. This may be followed by a discharge of pus containing staphylococcus aureus.

These well-known principles which are true of infection in general are also applicable to oral sepsis. Individuals with dental sepsis become sensitized to the protein of the infecting organisms. The protein of these organisms is to them no longer harmless and inert, but may under certain circumstances be poisonous to them. The protein of these organisms is thereafter a possible source of systemic disturbance. The sensitization produced is similar in every way to that observed in individuals with tuberculosis, furunculosis, etc., and the reactions which follow inoculation with killed bacteria or the reactions which follow inoculation with living bacteria by Nature as a result of the development of a new

infection are similar in every respect to those of the foregoing examples.

If an individual with oral sepsis develops an acute infection, such as tonsillitis or an acute alveolar abscess due to the same organism which may be infecting the gums, a general reaction follows, due to the action of the protein of these microorganisms upon the sensitized individual. A focal reaction in the gums may follow this and may cause a rapid increase in the inflammatory process there localized. It is possible for a focal reaction to occur even in lesions of the gums which have nearly healed and in this way cause recurrence of an apparently cured case of pyorrhea. Focal infections such as chronically infected tonsils, alveolar abscesses, etc., often harbor the same organisms as infected gums and often prevent permanent cure of oral sepsis through the activity of the bacteria of these localities giving rise to repeated focal reactions in the gum, and in this way causing repeated recurrence and extension of the local inflammatory process. The permanent cure of oral sepsis is, therefore, often dependent on the removal of all other sources of systemic infection. (See Figs. 31 to 36.)

In the previous paragraph the effect which coexisting infections may have upon oral sepsis has been dwelt upon. Oral sepsis may have an analogous effect upon all chronic foci of infection located elsewhere in the body which harbor the same microorganisms; for example, an acute alveolar abscess may cause a focal reaction in a chronically infected tonsil and give rise to an attack of acute tonsillitis. Likewise, it may cause a focal reaction in a chronically infected appendix, gall bladder or gastric mucosa and give rise to acute symptoms of local inflammation. The extraction of badly infected teeth may have the same effect, and may be followed in less than twenty-four hours by a severe, acute inflammatory reaction in distant organs (evidently a focal reaction).

So prompt an effect must be the effect of a reaction in the secondary focus followed by the rapid growth of organisms already there localized rather than to a fresh acute metastatic infection from the alveolar process. The following case illustrating this type of reaction is cited as a typical example:

Patient, aged sixty, relatively normal for her age, had ten infected teeth extracted. In less than twenty-four hours after this there developed a severe acute follicular tonsillitis. The infection several days later began to spread from the gums to the cheeks, pharynx, and tongue and gave rise to an extensive ulcerative stomatitis. A point worthy of emphasis in this case is the fact that the inflammatory reaction in the tonsils, while relatively distant from the gums, preceded by several days the infection of the neighboring tissues of the mouth. This attack of acute tonsillitis was analogous in every way to the acute inflammatory reaction in the lungs which may be brought about in tuberculous individuals by the injection of an overdose of tuberculin. It is by no means uncommon to observe pain in the region of the gall bladder, appendix, stomach, or joints a few hours after the extraction of teeth, and such may frequently be interpreted as focal reactions caused by the dissemination of microorganisms or their products from the alveolar process.

While extraction of the infected teeth frequently causes acute reactions in infected organs and occasionally in this way causes spread of disease, the good eventually derived usually far outweighs the ill. Frequently the clinical manifestations of chronic appendicitis, cholecystitis, gastric and duodenal ulcer, chronic arthritis, etc., clear up rapidly after the extraction of teeth even though the first effect may have been an increase in the inflammatory processes. Occasionally chronic inflammatory conditions which appear rather urgently to indicate the

need of surgery clear up after as simple a procedure as the extraction of a tooth. Even more gratifying is the relief occasionally obtained in chronically inflamed organs which can not be handled surgically, such as chronic nephritis and arrhythmia.

Combined with chronic inflammatory diseases of any organ there frequently exists a functional disturbance in the organ which appears out of all proportion to the gravity of the inflammatory lesion. This is without question frequently the effect of repeated focal reaction in the organ caused by activity in some distant focus. It is perhaps for this reason that uremia and other symptoms of renal insufficiency in cases of chronic intestinal nephritis, cardiac insufficiency, and arrhythmia in cases of chronic myocarditis, etc., are often relieved in a most gratifying way by the treatment of oral sepsis or other foci of chronic infection.

The sensitization of an infected individual against a given organism may be increased by repeated inoculations hypodermically with the protein of that organism. The same procedure may produce tolerance for the organism. Whether the effect produced is increased sensitiveness or increased tolerance is largely dependent upon the rate at which the inoculations are given and the size and rate of increase in the dose. If the dose is too large or too frequently repeated, the sensitiveness may be increased. If the dose is not larger than can be tolerated by the individual and is not repeated too frequently, a high degree of tolerance may be gained. If tuberculous individuals are inoculated with large and rapidly repeated doses of tuberculin, a marked increase in sensitiveness is produced and a rapid advance in the disease may follow. This practice in tuberculin therapy has given disastrous results in the past. If tuberculous individuals are repeatedly inoculated with carefully graduated doses of tuberculin at appropriate intervals, a tol-

erance may be developed which is so great that a quantity of tuberculin can eventually be given without deleterious effect which is thousands of times as great as that which would have originally caused a reaction. The toxic effect of given infection would seem, on the basis of experimental results and the above cited facts, to depend in part upon the degree of sensitization in the individual, in part upon the degree of tolerance which had been developed, and, finally, in part upon the rate and regularity with which the organisms and their products gain entry into the circulation. This explains the fact that some individuals with dental sepsis show many of its toxic effects while others similarly infected show relatively few.

In the preceding pages the toxic effect of bacteria and their products upon infected tissues has been dwelt upon. The effect which such products may have upon uninfected tissues is perhaps even more interesting, though perhaps of less clinical importance. This effect can be well illustrated by describing the effect upon animals of alien protein not of bacterial origin.

It is now well known that normal untreated guinea pigs may be inoculated with large amounts of horse serum or other alien protein without apparent immediate ill effect and that if this inoculation is repeated after a period of several weeks, the animal is likely to die in a short time with a peculiar syndrome of symptoms which has been called anaphylactic shock. The term anaphylaxis was introduced by Richet who looked upon the phenomena fallaciously as an effect of a lack of protection. The phenomenon had previously been observed by Magendi and Theobald Smith. Since then, Arthus, Vaughan, von Pirquet, Rosenau and Anderson, Meltzer, Auer and Lewis, and others have added interesting observations which have made the subject one of great general interest. The symptoms produced by treating sensitized animals with alien proteins vary in different animals,

vary with the degree to which the animal has been sensitized, and finally vary with the nature and quantity of the protein used. Guinea pigs are quite sensitive to the effect of alien proteins, and if a second dose is administered to them after an appropriate interval of time, death often ensues as a result of bronchiole constriction and asphyxia. In other animals different symptoms are dominant and often are of a relatively mild nature. The symptoms which may be expected in animals in more or less marked degree are as follows: irritability followed by depression, prostration and often apparent paralysis, dyspnea, discharge of urine and feces, lowering of blood pressure, a rise or lowering of body temperature, reduction in the leucocyte count, eosinophilia, delay in the coagulation time of the blood, agglutination of the blood platelets with lowering of the platelet count as a result of the larger clumps being filtered off by the capillaries, constriction of the bronchioles frequently giving rise to an embarrassment of respiration which in the more extreme cases prevents entirely the expulsion of air and causes extreme inflation of the lungs, contraction of nonstriated muscle generally giving rise to bronchiole spasm, increased peristaltic movements in the stomach and intestine, emptying of bladder and rectum, bloody diarrhea, etc., increased glandular secretion especially of the liver, pancreas, lacrimal, and salivary glands, increased irritability of the peripheral nerves, etc. In humans, anaphylactic symptoms occasionally follow the use of therapeutic sera, vaccines, etc. These are usually relatively mild as compared with those shown by sensitized animals. Those most frequently observed are urticaria, erythema, angioneurotic edema, pain and swelling of the joints, and leucopenia. The symptoms in humans may be much more severe, however, and may in some instances simulate the severe symptoms observed in animals. Very severe reactions are occasionally observed

after the use of pollen extracts in the treatment of hay fever and asthma.

Guinea pigs, which are highly sensitive to anaphylactic shock, may be sensitized by minute quantities of a foreign serum. One one-thousandth milligram or less often suffices to sensitize them to such a degree that a subsequent injection of $\frac{1}{20}$ c. c. or more, given after an incubation period of two or three weeks, almost invariably results in convulsions, constriction of the bronchioles and rapid asphyxia.

Guinea pigs may be sensitized to alien protein by methods other than that of subcutaneous inoculation; for example, they can be sensitized by the feeding of certain proteins, by the introduction of proteins through the skin by inunction, through the mucous membranes of the colon by rectal injection, through the mucous membrane of the respiratory tract by the use of sprays, etc. In fact, if an alien protein gains entry undigested into the circulation or tissues in any way, it may sensitize.

Proteins vary in the degree to which they can sensitize. Serums and egg albumens, for example, may produce a high degree of sensitization, whereas bacterial proteins usually produce relatively very little.

Protein sensitization as observed clinically in humans probably has many sources of origin. It is difficult to understand just how the sensitization takes place. Some individuals, for example, become sensitized to the protein of certain articles of food (egg, beef, strawberries, etc.), some to the pollen of plants (ragweed, golden rod, etc.), some to animal odors (horse, cat, guinea pig, etc.). The sensitization may reach such a degree that whenever the individual comes in contact even with minute amounts of the protein to which he is sensitized certain disagreeable and even dangerous symptoms of anaphylaxis are noticed, in fact, in many individuals almost infinitesimal quantities of the offending protein may produce severe

symptoms of anaphylaxis. Individuals sensitized to horse albumen may show symptoms of anaphylaxis after inhaling the infinitesimal quantity of horse protein in the air around horses. Individuals sensitized to egg albumen may have urticaria after eating minute quantities of egg albumen. Individuals sensitized against plant pollen may have hay fever or asthma after contact with the minute quantity of pollen in the air. Severe anaphylactic shock can be produced in such individuals by the subcutaneous injection of as small an amount as one one-thousandth milligram of an extract of the pollen to which they are sensitized.

The degree to which sensitization may develop in humans is almost unbelievable. I observed a patient who was so sensitized to cat protein that whenever she was in the room with a cat, symptoms of anaphylaxis were noticed. She told friends of this peculiarity. One disbelieving brought a kitten into the house in his pocket believing that if the patient were not aware of its presence, the symptoms would not appear. The effect on the patient was such, however, as to alarm the entire household and convince the friend that the experiment would not bear repetition. Kolmer mentions an instance of an individual so sensitized against rabbits, guinea pigs, and horses, that if the air of the room in which these animals were kept were passed fifteen minutes through lint and the lint extracted with saline solution, the application of this extract to the abraded skin of the individual would cause a marked local reaction. Protein sensitization acquired in nature as above described is greater in degree than that produced experimentally in animals or humans by the subcutaneous injection of a foreign protein. It is also of longer duration. Sensitization against plant pollen, for example, may last for years, whereas that produced artificially often diminishes rapidly after a few weeks.

The above examples which show the effect which may be produced in sensitized humans and animals by minute quantities of an alien protein are mentioned for the purpose of illustrating the extreme and diversified effect which may be produced under certain circumstances by small amounts of protein which to nonsensitive individuals are practically harmless. In pyorrhea pockets and alveolar abscesses the tissues of the human come in intimate contact with alien proteins and split proteins of many types and sources due to the activity of bacteria, protozoa, etc. The individual is not protected from the absorption of these by an intact epithelial membrane. It seems reasonable to presume that these occasionally gain entry into the tissues and blood and sensitize the individual in high degree and by virtue of this become possible sources of anaphylactic phenomena. Whereas bacterial proteins are not prone to cause the so-called anaphylactic phenomena experimentally, the fact that such may occur as an effect of bacterial products has been shown by the researches of Baldwin and Krause who working with the protein of the tubercle bacillus were able to produce in animals all the striking anaphylactic phenomena observed experimentally after the use of serums. In harmony with this is the well-known fact to be dwelt upon later that typical severe anaphylactic phenomena frequently complicate acute infectious diseases, such as rheumatic fever, tonsillitis, etc.

The work of Major is interesting in this connection. Working with doses of alien protein which were too small to cause visible anaphylactic phenomena in animals, he was able to produce profound metabolic disturbance and extreme emaciation believed to be due to a chronic anaphylactic state too mild to give rise to the usual visible manifestations of the condition. His inference drawn from the experiments that metabolic disturbances ob-

served clinically are often the result of a chronic mild anaphylactic state is in harmony with the fact that individuals often gain weight and strength after the treatment of chronic infections.

Numerous theories have been offered to explain protein sensitization. Those suggested by Vaughan, von Pirquet, and Wolff-Eisner are similar in the essential that it is assumed that an animal under the influence of an alien albumen develops enzymes which break it down and that the split products which result are poisonous and cause symptoms of intoxication. Vaughan, after exhaustive experimentation in this field, developed an elaborate theory which, as he suggests, may prove false in some respects, but which serves well the purposes of a working hypothesis. It is hardly possible in a work so brief as this one to go into the various details of the interesting and ingenious views of Vaughan, von Pirquet, Jobling, Novi, and others. For this, the reader is referred to the original contributions of these observers. (See Bibliography.)

A summary of the theories which have received most general acceptance is as follows: Albuminous materials taken as food are broken down into simpler compounds in the gastrointestinal tract by digestive enzymes and are rebuilt into the complex protein molecule during and after absorption. The protein molecule thus built up is distinctive for each species of animal. If the albuminous material of one species of animal gains entry into the circulation of another species without first being broken down by the digestive enzymes of the animal receiving it and then being rebuilt into its distinctive type of protein, the animal receiving it becomes sensitive to the protein of that particular species and remains so for a period of weeks or months or years. If, during this period of sensitization, the protein to which the animal has been sensitized is introduced into the circulation

in sufficient quantity, the animal may die in a few minutes of anaphylactic shock. If the animal survives, the symptoms of anaphylaxis usually disappear after a few hours. If the protein gains entry into the circulation in smaller quantity and repeatedly, the symptoms may be milder and simulate those observed as a result of acute or chronic infection.

It has been suggested that sensitization is due to the development of enzymes in the tissues of an animal for the purpose of destroying the foreign protein and that in breaking it down certain split products are formed which may be toxic and cause the severe symptoms observed in anaphylactic shock. The enzymes which are developed under the influence of a foreign undigested albumen are apparently remarkably specific and are able to digest only the type of protein which called them forth. The acquired faculty of animal tissues to furnish enzymes which are able to destroy alien protein of certain types and in so doing to liberate products which are poisonous to the animal constitutes the condition known as sensitization. The result of this is, that whenever a foreign protein to which an animal has become sensitized comes in contact with its tissues, it is broken down rapidly by its enzymes with the liberation of poisonous split products. If these are formed in quantity larger than the animal can tolerate, the symptoms of anaphylaxis, of varying severity result. As mentioned by Vaughan, the above conclusions may prove false in some respects, but at present serve the purposes of a working hypothesis. Other ingenious theories have been suggested, but it is hardly possible in this space to discuss them.

Tolerance for alien proteins may be developed in sensitized animals just as tolerance against tuberculin may be developed by tuberculous individuals. If an animal sensitized to horse albumen is given subcutaneously a dose of horse serum which produces an anaphylactic re-

action but which does not cause death, the animal becomes refractory for a certain period of time. Inoculation with horse serum during such a period has less than its usual deleterious effect. Tolerance for a given protein may be enormously increased in a sensitized animal or individual by giving subcutaneously graduated doses of the protein at appropriate intervals. Upon this principle depend the good results obtained in the treatment of hay fever with graduated doses of pollen extract and of horse asthma with graduated doses of horse protein.

Anaphylaxis as produced experimentally by animal inoculation is usually acute and of relatively short duration. Anaphylaxis as observed clinically in humans, however, is often chronic and of a milder nature; for example, bronchial asthma, first mentioned by Meltzer as an anaphylactic phenomenon, may continue for months, in fact may continue so long as the individual comes in contact with the protein to which he is sensitized. Also urticaria due to sensitization against egg albumen may continue for months or years unless the individual excludes egg from his diet.

As previously mentioned, bacterial proteins are not prone to cause anaphylactic phenomena upon injection into sensitized animals. Anaphylaxis may, without question, occur in humans, however, as a result of infection; for example, angioneurotic edema, urticaria, and erythema, and certain eczemas unquestionably anaphylactic in nature occasionally occur during an attack of acute articular rheumatism, tonsillitis and other infectious diseases. Such phenomena are occasionally observed also in association with chronic foci of infection such as chronically infected tonsils and alveolar abscesses and the same may clear up immediately or soon after the offending foci are removed.

The problem of interest in this connection is not the question whether or not oral sepsis is a possible cause

of anaphylactic phenomena, for one is safe in assuming that this is so. The question of interest is whether or not oral sepsis is a frequent source of anaphylactic phenomena, and whether or not this is a factor of clinical importance in the causation of functional or organic disease. The fact that the areas of sepsis are small is not inimical to the assumption that such is the case, for severe anaphylactic reactions may be caused by traces of a foreign protein. The fact that it is probably a relatively frequent and important source seems likely from the fact that oral sepsis is a chronic disorder of years' duration. It harbors organisms of many types and finds the body repeatedly in varying states of sensitization and tolerance.

According to the views of Vaughan, alien proteins have affinities for certain tissues just as do certain strains of bacteria, and exert their toxic effect chiefly upon the tissues with which they combine. Whether the effect be one of functional disturbance or of demonstrable organic lesion would appear to depend upon the severity and duration of the intoxication. Just how great a factor such toxic albumens derived from the alveolar process are in themselves a cause of organic changes in normal uninfected tissues is, of course, a matter of conjecture. It seems to be, however, a frequent cause of functional disorder. The following symptoms, which are rather difficult to classify, appear frequently to be a result in part at least of protein intoxication and often can be relieved by the removal of infected teeth or of other sources of chronic infection.

General symptoms, such as nervousness, malaise, dizziness, drowsiness, inability to concentrate, inexplicable weakness, prostration after slight mental or physical exertion, headache made worse by eyestrain, or mental or physical exertion, slight fever or subnormal temperature, slight tachycardia or bradycardia, instability of the vaso-

motor center, etc., are produced occasionally in sensitized individuals by the use of vaccines made from cultures taken from infected gums and are often markedly relieved by the treatment of chronic infections.

Angioneurotic edema, urticaria, erythema multiforme, and certain types of eczema, according to Sutton, may be anaphylactic in origin and often clear up rapidly after the removal of periodontal or other chronic infections. We have observed each of the above mentioned lesions appear during treatment with autogenous vaccines and pollens and disappear as soon as treatment was discontinued.

Disturbances in the function of organs supplied by the vegetative nervous system are frequently observed as a result of oral sepsis. Pottenger has observed both vagotonic symptoms and symptoms due to hypertonus in the sympathetic system as a result of chronic tuberculosis, the former being the more common in latent cases. The disturbances most commonly observed as a result of oral sepsis simulate the vagotonic, and may give rise when combined with other contributing factors to such conditions as asthma, motor and secretory neuroses of the stomach and intestine, such as gastric hyperacidity, hyperperistalsis and spasticity, mucous colitis, chronic diarrhea, spastic constipation, etc., functional disturbances of the kidney, bladder, and sexual organs. Such conditions often clear up rapidly after the treatment of chronic infection foci.

Combined with organic disease in any organ, there may be functional disturbance which is out of all proportion to the gravity of the anatomic lesion. Emotions, abnormal reflexes, or intoxications of various sorts, anaphylaxis, etc., may add greatly to disordered function in any organ. Often symptoms of organic disease are made worse by products absorbed from chronic areas of infection such as oral sepsis, and perhaps it is for this

reason the symptoms of organic disease, which may or may not be infectious in origin, are often strikingly ameliorated by the treatment of infections which may bear no apparent relation to them. The symptoms of exophthalmic goiter, renal insufficiency, uremia, Addison's disease, epilepsy, chronic myocardial insufficiency, cardiac arrhythmia, arterial hypertension, etc., are often benefited in marked degree by the proper handling of chronic infections.

Finally, as previously mentioned, oral sepsis or chronic infection from any source may exert an untoward influence upon the course of other nonrelated infections and increase the functional disturbance caused by them. Acne, furunculosis, general paresis, tabes dorsalis, tuberculosis, etc., are often more amenable to treatment and their systemic effect is less marked if all nonrelated sources of infection are given the attention they deserve.

Oral sepsis as a source of systemic intoxication is not the cause of all ills. It is, however, a direct cause of many ills, and also frequently adds to the ill effect of other diseases.

CHAPTER VII

HEADACHE RELATED TO ORAL SEPSIS

Chronic infections, especially those in the alveolar process are relatively frequent causes of chronic headache. Many individuals who have headache after eyestrain may discard their glasses after the treatment of chronic infection. This is not apparently due to a relationship between infection and eyestrain, but rather to the fact that eyestrain which so frequently figures in the etiology of headache is not always a primary or sole cause, but often is simply a contributory cause. Oral sepsis, infected tonsils, infected ethmoids, hyperacidity due to chronic appendicitis or gallstones are directly or remotely the cause of headache more often perhaps than eyestrain. Defective teeth may be responsible for headache in several different ways.

First, they may be sources of arthritis in the cervical spine, of myositis in the muscles of the neck, or of inflammatory processes in the bursæ, in the tendon sheaths, or at the points of attachment of the tendons to the skull and cervical vertebræ. All the above may give rise to pain in the back of the neck reflected upward over the skull. This type of headache is common in individuals with postural defects,—the abnormal strain on the muscles, tendons, and ligaments of the neck being a factor perhaps in determining this localization for an arthritis. Such headaches may be constant for days, may be made worse or brought on by mental or physical exertion, fatigue, excitement, worry, eyestrain, indulgence in alcohol; in fact, by any condition which may act as an additional strain on the individual. The pain may be so severe as to interfere with business activity or pleasure.

When headache such as the above has its origin in oral sepsis or other infections, relief can often be secured by their treatment. If the condition is of long standing, and a considerable degree of rigidity of the cervical spine has resulted, the immediate effect is not so striking. Apparently headaches giving symptoms such as those above described may be the result solely of a toxic effect of sepsis, at least, it is frequently not possible even in cases of headache of years' duration to demonstrate by physical examinations or by x-ray, the usual manifestations of local inflammatory processes at the base of the skull or in the cervical vertebræ. The fol-

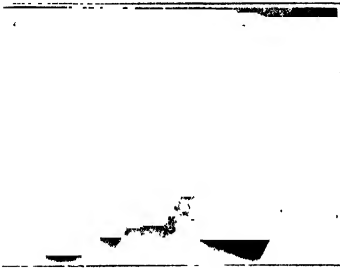


Fig. 166.



Fig. 167.

Figs. 166-167.--Roentgenograms showing broaches which had been introduced with case through the root canal and alveolar process practically as far down as the dental nerve. It shows the case with which infection and pressure from an alveolar abscess can be transmitted to the dental nerve and cause neuritis or neuralgia.

lowing case is cited as a typical example of headache due primarily to dental sepsis.

Patient, female, age twenty-five, had for five years been subject to headache starting in back of neck and radiating over the skull, lasting usually for several days at a time and often being so severe as to render patient incapable of mental or physical exertion. Such attacks could be brought on by exertion, eyestrain, fatigue, by the use of alcohol, or by indigestion. The condition was definitely alleviated by the removal of the tonsils. Later

several root abscesses were discovered and treatment of these was advised. The treatment of each abscess was followed by such severe headache as to confine the patient to bed (probably a focal reaction). Since completion of dental work, however, the patient has been completely relieved.

Second, oral sepsis may be a source of neuralgia or neuritis in any or all of the branches of the facial nerve.



Fig. 168.

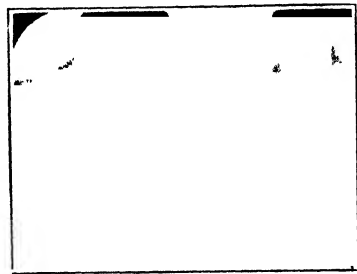


Fig. 169.



Fig. 170.

Fig. 168.—Case of severe headache due to cervical arthritis and myositis which was completely, and apparently permanently relieved by the extraction of the tooth shown in the above roentgenogram.

Fig. 169.—Case of severe facial neuralgia relieved by extraction of the tooth shown in the above roentgenogram.

Fig. 170.—Case of headache due to antrum infection, the source of which was an abscess at the root of a bicuspid tooth. A probe could be introduced into the antrum.

Tic douloureux is not included here, since this condition occurs frequently in individuals whose teeth have all been extracted.

Third, headache is occasionally a referred toothache. Frequently a patient not only fails to localize the par-

ticular tooth which causes pain, but occasionally can state simply that the pain is localized somewhere in the head. Such a toothache may be so severe as to suggest brain tumor. A tooth need not be abscessed to cause such pain and occasionally can be found only after a most careful examination by a dentist. The following is an example of such a case:

Patient, male, age forty-three, complained of a constant and severe headache of about three weeks' duration. It interfered with sleep and caused loss of weight and was at times so severe as to cause the patient to cry out. Physical, laboratory, and roentgen examinations were negative throughout except for disclosing a pulp stone in a first upper molar tooth. This tooth on examination by Dr. R. M. Siebel showed the pulp of one root to be diseased. Two of the roots were vital. The tooth was cocainized and the pain disappeared immediately. Following extraction of the tooth, the headache was permanently relieved.

Fourth, an alveolar abscess may rupture into the antrum of Highmore and cause headache. The local symptoms may be so slight as to escape notice entirely. The following is a typical example:

Patient, woman, age forty, had been subject to headaches since about the age of eighteen, at which time a bicuspid tooth had been devitalized. Headache resisted all therapeutic measures except the strongest sedatives. Examination showed an abscessed bicuspid tooth. When the tooth was extracted a probe could be introduced into the antrum. Following the extraction of the tooth, there was a marked exaggeration of the headache for some time, and a profuse nasal discharge evidently due to an exacerbation of the antrum infection. Following this, treatment was directed to the diseased antrum, and since then the patient has been relatively free from headache.

CHAPTER VIII

SUMMARY AND CONCLUSIONS

It has been the purpose in this volume to assemble facts which show the many ways in which chronic sepsis of any variety may be a source of systemic ill, and that, of all chronic active infections, those which occur at the roots of the teeth are perhaps the most common. The development of this knowledge marks a great practical advance in the diagnosis and treatment of disease. It is now apparent that many disorders, which in previous years were considered obscure in origin and incurable, are due wholly or in part to chronic infection, and that such diseases can often be cured or retarded in their progress by removal of the primary source of infection. It is also apparent that the occurrence of chronic sepsis is far more frequent than was formerly supposed. It is so frequent, in fact, that few of mature age are wholly free from it.

Tabulations of one thousand medical cases which we have examined in consulting office practice on account of miscellaneous systemic complaints gave the following results: Sources of chronic or frequently recurrent infection were found in ninety-seven per cent (this included active tuberculosis, syphilis, chronic gastrointestinal disorders due to abnormalities in the appendix, gall bladder, to gastric and duodenal ulcer, extreme ptosis, adhesions, chronic infection in the respiratory genitourinary tract, etc.). A marked degree of oral sepsis was found in roentgenograms in 66 per cent. In 34 per cent either none was found or the amount was so slight that many would consider it negligible from the standpoint of systemic ill. Diseased tonsils appeared to be possible sources of

disease in a large percentage of cases. Sixteen per cent of the cases gave a history of chronic nasal discharge or showed deviation of the septum, spurs, polypi, or other abnormal conditions in the nose. In a relatively small percentage of these there was tenderness over the frontal or ethmoid sinuses or abnormal opacities in the region of the sinuses disclosed by x-ray. Sixteen per cent of the cases had digestive disturbance or other symptoms associated with tenderness in the right iliac fossa. A minority of these were thought to have an abnormal appendix. Seven per cent had symptoms which appeared to be attributable to the gall-bladder. In addition a relatively large percentage had digestive disturbance due to either appendix or gall-bladder disease, which could not be definitely diagnosed, or to adhesions, extreme ptosis, gastric or duodenal ulcer, etc. Eleven per cent gave positive Wassermann tests. (Bloods sent in because of suspected syphilis were excluded from these statistics.) In over half of these (6 per cent of the total number of cases examined) syphilis of the central nervous system was suspected clinically and verified by lumbar puncture. In 7 per cent the presence of active tuberculosis was positively demonstrated. A miscellaneous group of infections was found in a large number of cases. The great majority of cases had more than one chronic infection. Of the 66 per cent having a considerable degree of oral sepsis, 72 per cent had other chronic infections in addition. In but 18 per cent was oral sepsis the only active infection found.

The frequent occurrence of oral sepsis, the frequency with which other infections coexist and the frequency with which patients give a history of having had one or more of the more severe acute infectious diseases, make it difficult or impossible from statistics alone to determine the exact role which oral sepsis plays in the etiology of systemic disease. It is interesting, how-

ever, to compare the statistics of Thoma with those here presented. In examining patients in the Robert B. Brigham Hospital, where practically all the inmates are sufferers from chronic disease, Thoma found alveolar abscesses in 88 per cent. This ratio is higher than that which we found to have oral sepsis in marked degree (66 per cent) in ambulatory patients observed in office practice. E. E. Irons reports that of 124 cases examined with miscellaneous diseases, 44 per cent had alveolar abscesses. Of these the arthritic group showed abscesses in 76 per cent. Of the nephritic and cardiovascular group 47 per cent had abscesses. Of the miscellaneous group, not rheumatic in origin, only 23 per cent had oral sepsis. This is less than one-third the percentage found in the arthritic group.

Among individuals examined by A. D. Black, without reference to complaint, the percentage of periodontal infections was 56 for persons under twenty-five years of age, 72 for those between the ages of twenty-five and thirty years, 87 for those between thirty and forty years, 89 for those between forty and fifty, and 100 per cent for individuals over fifty years of age.

There is, of course, some variation in statistics reported by different observers. This is due in part to the class and age of the patients examined and in part to slight differences in the basis upon which the diagnosis of sepsis is made.

Facts which seem certain from evidence now at hand may be summarized as follows: Dental sepsis is one of the commonest of the chronic active infections and for this reason ranks theoretically as a very frequent cause of ill health. The ill effects of the lesions frequently appear clinically to be out of all proportion to their size, a fact which may be explained by their frequent occurrence in the form of blind abscesses, by their frequent localization in osseous tissue, which allows no expansion,

and, finally, by the frequency with which the infected areas are exposed to pressure transmitted by the teeth during mastication.

Some individuals with small areas of sepsis may show many serious systemic ill effects, while others with greater amounts may apparently suffer less.

Oral sepsis as a focus of chronic active infection may be a source of ill health in many different ways. It may harbor and distribute organisms which, under certain conditions, may infect other tissues and give rise to acute or chronic inflammatory lesions. It may have a toxic effect with ensuing disease in both normal and diseased organs. This effect in healthy individuals may perhaps be slight. It may be decidedly harmful, however, in individuals who are depleted by disease, injurious habits, overwork, or age. It may favor the advance of infectious diseases due to organisms distributed from the alveolar process as a primary source of infection and may also favor the advance and augment the symptoms of other infections which are in no wise related to it. It may also cause functional disturbance in relatively normal organs by furnishing an alien protein to which an individual may have become highly sensitized. In the same way it may increase functional disturbances due primarily to organic disease. Finally, it may cause local pain, referred pain, and headache.

The immediate therapeutic result which follows the eradication of oral sepsis varies and the more conservative often hesitate to promise too much. Frequently the result is excellent. On the other hand, it is often disappointing. It is likely to be disappointing if the removal has been incomplete, or if other coexisting infections have been left intact, also if extreme anatomic changes have been brought about as a result of chronic systemic infection. The best results are obtained in those instances where the systemic effects are chiefly toxic. In

these cases a brilliant result often can be secured within a short time.

The treatment of oral sepsis is largely a dental problem, and is at present the subject of much interesting investigation. Several factors must be considered in the choice of method, namely, adequacy and permanence of result, possibility of restoring surfaces for mastication, time and expense to the patient. Time and expense often make extraction of teeth with damaged roots the method of preference. In addition to this, it is the safest and surest means of eradicating sepsis. Frequently conservative methods are justified, however, especially if the resulting systemic diseases are not extremely serious and if there exists no constitutional disorder such as diabetes, severe anemia, etc., which may lower resistance to infection and decrease the likelihood of success.

The treatment of oral sepsis may be divided into three classes: first, treatment of existing sepsis; second, treatment of defective teeth for the purpose of preventing sepsis; third, treatment of children with the view of obtaining perfect development of the teeth and oral cavity, thereby avoiding some of the various defects which eventually lead to sepsis. The last method naturally is the best. Eventually it is also the cheapest and easiest. Under ideal conditions prophylaxis should begin in early childhood and should include the proper handling of every abnormal condition of the mouth, throat, and nose which may give rise to mouth-breathing, debility, infection, etc., and in this way interfere with the proper development in the architecture of the throat, nose, jaw, and teeth. If the public, as well as physicians and dentists, were aware of the serious influence which defective teeth have on the development and health of the average individual, oral prophylaxis would hold the important place in preventive medicine that it so richly deserves, and the result would be economy in time and ex-

pense to the patient, increased physical and mental efficiency, a greater average duration of life, better preservation of the tissues in old age, and fewer chronic diseases.

BIBLIOGRAPHY.

- Adami, J. G.: Adaption and Disease, *Brit. Med. Jour.*, 1917, i, 837, 872; *ibid.*, 1917, ii, 9.
- Parenteral Digestion and Immunity, *Canad. Med. Assn. Jour.*, 1915, v, 569.
- Anders, J. M.: Mouth Sepsis, *Jour. A. M. A.*, 1917, lxxviii, 567.
- Oral Sepsis, *New York Med. Jour.*, 1917, cv, 433.
- Arthus, M.: Injections répétées de sérum de cheval chez le lapin, *Compt. rend. Soc. de biol.*, 1903, lv, 817.
- Auer, J.: The Effects of Resection of One Vagus upon Serum Anaphylaxis in Guinea Pigs, *Proc. Soc. Exper. Biol. and Med.*, 1910, vii, 103.
- The Physiology of the Immediate Reaction of Anaphylaxis in the Guinea Pig, *Jour. Exper. Med.*, 1910, xii, 151.
- Auer, J., and Lewis, P. A.: Acute Anaphylactic Death in Guinea Pigs, *Jour. A. M. A.*, 1909, liii, 458.
- Babcock, R. H.: Focal Mouth Infections: Their Systemic Effect and Treatment, *N. Am. Therap. Soc.*, 1916, p. 129.
- Baehr, G.: Glomerular Lesions of Subacute Bacterial Endocarditis, *Jour. Exper. Med.*, 1912, xv, 330.
- Baldwin, E. R.: Studies on the Tuberculin Reaction, *Studies of the Saranac Laboratory for the Study of Tuberculosis, 1900-1904*, Boston, 1905.
- Bass, C. C., and Johns, F. M.: Pyorrhea Dentalis and Alveolaris, *Jour. A. M. A.*, 1915, lxiv, 553.
- Beck, J. C.: Chronic Focal Infection of the Nose, Throat, Mouth, and Ear, *Jour. A. M. A.*, 1914, lxiii, 1636.
- Bellei, G.: A Short Contribution to the Study of General Infection Produced by *Staphylococcus Aureus* and by the *Streptococcus*, *Lancet*, 1902, i, 807.
- Berger, A.: Dento-alveolar Abscess, Items of Interest, 1914, xxxvi, 641.
- Billings, F.: Chronic Focal Infections and Their Etiologic Relations to Arthritis and Nephritis, *Arch. Int. Med.*, 1912, ix, 484.
- Chronic Focal Infections as a Causative Factor in Chronic Arthritis, *Jour. A. M. A.*, 1913, lxi, 819.
- Chronic Infectious Endocarditis, *Arch. Int. Med.*, 1909, iv, 409.
- Clinical Aspect and Medical Management of Arthritis Deformans, *Ill. Med. Jour.*, 1914, xxv, 11.
- Focal Infection, *Jour. A. M. A.*, 1916, lxxvii, 847.
- Focal Infection, New York, Appleton and Co., 1917.
- Focal Infection: Its Broader Application in the Etiology of General Disease, *Jour. A. M. A.*, 1914, lxiii, 899.
- Mouth Infections as a Source of Systemic Diseases, *Jour. A. M. A.*, 1914, lxiii, 2024.
- Black, A. D.: Ocular Diseases Resulting from Dental Lesions, *Ophth. Rec.*, 1915, xxiv, 610.
- Roentgenographic and Microscopic Studies of Tissues Involved in Chronic Mouth Infections, *Jour. A. M. A.*, 1917, lxix, 599.
- Studies of the Investing Tissues of the Teeth as a guide for Treatment of Chronic Alveolar Abscesses and Suppurative Pericementitis, *Jour. Allied Dent. Soc.*, 1917, xii, 1917.

- The Diseases and Treatment of the Investing Tissues of the Teeth, Berkeley, 1916.
- Black, G. V.: *Special Dental Pathology*. Chicago, Medico-Dental Publishing Co., 1915.
- Blair, V. P.: *Dental Disorders and Peridental Infections: Their Relation to Neighboring Organs*, Surg., Gynec. and Obst., 1914, xviii, 941.
- Blake, F. G.: The Classification of Streptococci, *Jour. Med. Research*, 1917, xxxvi, 99.
- Bloodgood, J. C.: The Preexisting Lesions in the Oral Cavity and Their Relation to Malignant Disease, *Dental Rev.*, 1917, xxxi, 359.
- Bramwell, B.: Notes on the Treatment of Pernicious Anemia, *Brit. Med. Jour.*, 1909, i, 209.
- Branson, C. B.: Prevention of and Immunity from Dental Diseases, *Dental Summary*, 1917, xxxvii, 259.
- Brooks, E.: Susceptibility to and Immunity from Caries, *Dental Summary*, 1916, xxxvi, 941.
- Brophy, T. W.: Oral Infections, *Med. Clin.*, Chicago, 1916, ii, 133.
- Brown, T. R.: The Relationship between Diseases of the Mouth and General Diseases, *Jour. Nat. Dental Assn.*, 1917, iv, 866.
- Bunting, R. W.: The Pathology of the Dental Pulp, *Brit. Dental Jour.*, 1915, xxxvi, 409.
- Bush, B. E.: The Close Relation of the Dentist and the Physician, *Jour. A. M. A.*, 1910, lv, 752.
- Butt, W.: Nose, Throat, and Ear as Neighboring Organs to the Teeth, *Dental Cosmos*, 1915, lvii, 837.
- Cabot, R. C.: Analysis of Six Hundred Cases of Heart Disease, *Jour. A. M. A.*, 1914, lxiii, 1461.
- Castro, T. D.: Care of Focal Infection from the Dental Standpoint, *Therap. Gaz.*, 1917, xxxiii, 3, 5.
- Colyer, J. E.: *Dental Disease in Its Relation to General Medicine*, London, Longman's, 1911.
- Oral Sepsis and Its Relation to General Diseases, *Jour. Brit. Dental Assn.*, 1902, xxiii, 409.
- Cook, R. A.: Protein Sensitization in the Human with Special Reference to Bronchial Asthma and Hay Fever, *Med. Clinics N. Am.*, 1917, i, 721.
- Craig, C. B.: Periodontal Infection as a Causative Factor in Nervous Diseases, *Jour. A. M. A.*, 1914, lxiii, 2027.
- Crowe, S. J., Watkins, S. S., and Rothholz: Relation of Tonsillar and Nasopharyngeal Infections to General Systemic Disorders, *Bull. Johns Hopkins Hosp.*, 1917, xxviii, 1.
- Daly, R. R.: Relations of Dental and Rhinologic Work, *Dental Cosmos*, 1915, lvii, 43.
- Darling, B. C.: Oral Sepsis as a Focus of Infection, Its Bacteriological, Dental, and Roentgenological Aspects, *Am. Jour. Roentgenol.*, 1916, iii, 158.
- Davis, D. J.: Bacteriological and Experimental Observations on Focal Infection, *Arch. Int. Med.*, 1912, ix, 505.
- Bacteriology and Pathology of the Tonsils and Especial Reference to
• Chronic Articular Renal and Cardiac Lesions, *Jour. Infect. Dis.*, 1912, x, 148.
- Davis, W. T.: The Interrelation of the Teeth and the Eye, *Dental Cosmos*, 1915, lvii, 769.
- De Vecchis, B.: New Problems Regarding Tubercular Infection and a Special Treatment for Cervical Adenitis Following Oral Sepsis, *Dental Cosmos*, 1915, lvii, 737.
- Dick, G. F., and Dick, G. R.: The Bacteriology of the Urine in Non-suppurative Nephritis, *Jour. A. M. A.*, 1915, lxv, 6.

- Dorrance, G. M.: Enlarged Cervical Glands, with Special Reference to the Mouth as an Etiological Factor, *Dental Cosmos*, 1913, lv, 808.
- Doxtater, L. W.: Constitutional Infection Due to Chronic Dento-alveolar Abscess and Pyorrhea Alveolaris, *Dental Register*, 1915, lxix, 470.
- Eisen, E. J., and Ivy, R. H.: Roentgenologic Examination in Elimination of the Mouth as a Source of Infection in Systemic Disease, *Am. Jour. Roentgenol.*, 1916, iii, 269.
- Eppinger, H., and Hess, L.: *Vagotonia*, Trans., ed. 2, New York Nervous and Mental Disease Publishing Co., 1917.
- Eustis, R. S.: Endocarditis in Children, *Boston Med. and Surg. Jour.*, 1915, clxxiii, 348.
- Fisk, E. L.: The Role of Mouth Infections and Mouth Abnormalities in the Causation of Disease, *Internat. Jour. Orthodontia*, 1916, ii, 693.
- Freundlich, D. B.: The Teeth as a Primary Factor in Diseases of the Ear, Nose, and Throat, *Laryngoscope*, 1915, xxv, 40.
- Gay, F. P., and Southard, E. E.: On the Mechanism of Serum Anaphylaxis and Intoxication in the Guinea Pig, *Jour. Med. Research*, 1908, n. s., xiii, 407.
- Gies, W. J., and collaborators: Studies of Dental Caries, with Special Reference to Internal Secretions in Their Relation to the Development and Condition of Dental Enamel, *Proc. Soc. Exper. Biol. and Med.*, 1917, xv, 5.
- Gilmer, T. L.: Chronic Oral Infections, *Arch. Int. Med.*, 1912, ix, 499.
- Gilmer, T. L., and Moody, A. M.: A Study of the Bacteriology of Alveolar Abscess and Infected Root Canals, *Jour. A. M. A.*, 1914, lxiii, 2023.
- Goldberg, H. A.: Oral Infection and Its Relation to Arthritis, *Med. Rec.*, 1917, xci, 185.
- Pyorrhea Alveolar, Alveolar Abscess and Their Relations to Arthritis, Items of Interest, 1916, xxxviii, 651.
- Grievess, C. J.: Artificial Production of Apical Necrosis by Root Canal Drugging, *Dental Cosmos*, 1915, lvii, 1119.
- Dental Periapical Infections as the Cause of Systemic Disease, *Dental Cosmos*, 1914, lvi, 52.
- Systemic Pus Poisoning Associated with Diseased Dental Apical Regions, Items of Interest, 1911, xxxiii, 339.
- Secondary Infections, *Jour. Allied Dental Soc.*, 1914, ix, 178.
- The Relation of the Internal Secretory Organs to Malocclusion, Facial Deformity, and Dental Disease, Items of Interest, 1915, xxxvii, 661.
- The Relation of the Vitality of the Periapical Cementum and Adjacent Tissues to the Patient's Health, and the Status of the Dental Profession, *Dental Cosmos*, 1915, lvii, 1112.
- The Responsibilities of the Dentist in Systemic Diseases Arising from Dento-alveolar Abscess as Illustrated by the Etiology of Peridental Abscess, *Dental Cosmos*, 1914, lvi, 564.
- Unhygienic Mouths, *Dental Cosmos*, 1913, lv, 1102.
- Hamman, L. V., and Wolman, S.: *Tuberculin in Diagnosis and Treatment*, New York, Appleton and Co., 1912.
- Hartzell, T. B.: Report of the Minnesota Division of the Scientific Foundation and Research Commission, *Jour. Nat. Dental Assn.*, 1915, vii, 333.
- Report of the Mouth Infection Research Corps of the National Dental Association for Year Ending July, 1914, *Off. Bull. Nat. Dental Assn.*, 1914, i, 48.
- Secondary Infections Having Their Primary Origin in the Oral Cavity, *Jour. Allied Dental Soc.*, 1914, ix, 166.
- Some Evidences of the Importance of the Dental Path as a Source of Serious Localized and General Infections, *Jour. Nat. Dental Assn.*, 1916, iii, 172.

- The Clinical Type of Arthritis Originating About the Teeth, Jour. A. M. A., 1915, lxxv, 1093.
- The Mouth as a Factor in the Pathogenesis of Heart, Kidney, and Joint Inflammation, Jour.-Lancet, 1910, xxxvi, 215.
- Two Preliminary Reports of Arthritis Caused by Dental Abscess, Off. Bull. Nat. Dental. Assn., 1914, i, 4.
- Hartzell, T. B., and Henrici, A. T.: The Dental Path: Its Importance as an Avenue to Infection, Surg., Gynce. and Obst., 1916, xxii, 18.
- The Pathogenicity of Mouth Streptococci and Their Role in the Etiology of Dental Diseases, Jour. Nat. Dental. Assn., 1917, iv, 477.
- Henrici, A. T.: The Specificity of Streptococci, Jour. Infect. Dis., 1916, xix, 572.
- Holman, W. L.: The Invasive Quality of the Streptococci in the Living Animal, Am. Jour. Med. Sc., 1917, cliii, 427.
- Howe, P. R., and Hatch, R. E.: A Study of Microorganisms of Dental Caries, Jour. Med. Research, 1917, xxxvi, 481.
- Hoxic, G. H.: Pyorrhoea Due to Organisms Other than the Amebas, Jour. A. M. A., 1915, lxxv, 1908.
- Hudnut, P. A.: Mouth Infections in Their Relation to Systemic Disease, Boston Med. and Surg. Jour., 1917, clxxvi, 695.
- Hunter, W.: Pernicious Anaemia, London, C. Griffin and Co., 1901.
- Huschart, J. H.: The Relation Diseases of the Teeth Bear to the Eye and Ear, Dental Brief, 1907, xii, 799.
- Irons, E. E.: Dental Infections and Systemic Disease; Treatment and Results, Jour. A. M. A., 1916, lxxvii, 850.
- Jackson, L.: Experimental Rheumatic Myocarditis, Jour. Infect. Dis., 1912, xi, 240.
- Jobling, J. W., Petersen, W. F., and Eggstein, A. A.: The Mechanism of Anaphylactic Shock, Studies on Ferment Action, Jour. Exper. Med., 1914, xx, 37; *ibid.*, 1915, xxii, 401.
- Johnson, A. G.: Mouth Infections as a Factor in Prolonging Disability in Trauma, Railway Surg. Jour., 1916-17, xiii, 292.
- Kaskin, W. H.: Oral Infections as a Result of Neglect During Childhood, Internat. Clin., 1915, 25, S., iv, 168.
- Klotz, O.: Experimental Bacterial Interstitial Nephritis, Tr. Assn. Am. Physicians, 1914, xxix, 49.
- Langworthy, H. G.: Some Remarks on the Removal of Troublesome Tonsils of Interest to Dentists, Dental Cosmos, 1913, lv, 718.
- Laroche, G., Riehet, C., and Saint-Girons, F.: L'anaphylaxie alimentaire, Paris méd., 1913-14, xiii, 485.
- L'anaphylaxie alimentaire aux oeufs, étude expérimentale, Arch. de méd. exper. et d'anat. path., 1914, xxvi, 51.
- LeCount, E. R., and Jackson, L.: The Renal Changes in Rabbits Inoculated with Streptococci, Jour. Infect. Dis., 1914, xv, 389.
- Lee, R. I.: Preventable Heart Disease, Boston Med. and Surg. Jour., 1915, clxii, 157.
- Lescobier, A. W.: The Bacterial Findings and Their Relationship to Pyorrhoea Alveolaris and Interstitial Gingivitis, Jour. A. M. A., 1917, lxxviii, 414.
- Lewis, P. A.: The Induced Susceptibility of the Guinea Pig to the Toxic Action of the Blood Serum of the Horse, Jour. Exper. Med., 1908, x, 1.
- Libman, E.: A Study of the Lesions of Subacute Bacterial Endocarditis with Peculiar Reference to Healing of Healed Lesions, with Clinical Notes, Am. Jour. Med. Sc., 1912, cxliv, 313.
- Clinical Features of Cases of Subacute Bacterial Endocarditis That Have Spontaneously Become Bacteria-Free, Am. Jour. Med. Sc., 1913, cxlvi, 625.

- Libman, E., and Celler, H. L.: The Etiology of Subacute Infective Endocarditis, *Am. Jour. Med. Sc.*, 1910, cxi, 516.
- Lowdermilk, R. C.: Hay Fever, *Jour. A. M. A.*, 1914, lxiii, 141.
- Mayo, C. H.: Mouth Infection as a Source of Systemic Disease, *Jour. A. M. A.*, 1914, lxiii, 2025.
- Major, R. H.: The Production of Acute and Chronic Kidney Lesions with *Bacillus Mucosus Capsulatus*, *Jour. Med. Research*, 1917, n. s., xxxii, 125.
- The Production of Kidney Lesions with *Staphylococcus Aureus* Toxins, *Jour. Med. Research*, 1917, n. s., xxx, 349.
- Milne, L. S.: Chronic Arthritis, *Jour. A. M. A.*, 1914, lxii, 593.
- Moody, A. M.: Lesions in Rabbits Produced by *Streptococci* from Chronic Alveolar Abscesses, *Jour. Infect. Dis.*, 1916, xix, 515.
- Moorehead, F. B.: The Prevalence of Chronic Mouth Infections and Their Management, *Jour. A. M. A.*, 1916, lxvii, 845.
- Meltzer, S. J.: Bronchial Asthma as a Phenomenon of Anaphylaxis, *Jour. A. M. A.*, 1910, lv, 1021.
- Navy, F. G., (et al): Anaphylatoxin and Anaphylaxis, *Jour. Infect. Dis.*, 1917, xx, 499, 536, 566, 589, 618, 629, 657, 776.
- Navy, F. G., and DeKruif, P. H.: Anaphylatoxin and Anaphylaxis, *Jour. A. M. A.*, 1917, lxviii, 1524.
- Nichols, E. H., and Richardson, F. L.: Arthritis Deformans, *Jour. Med. Research*, 1909, n. s., xvi, 149.
- Opie, E. L.: On the Relation of Combined Intoxication and Bacterial Infection to Necrosis of the Liver, Acute Yellow Atrophy and Cirrhosis, *Jour. Exper. Med.*, 1910, xii, 367.
- Osborne, O. T.: The Menace of Mouth Infections, *Jour. A. M. A.*, 1917, lxix, 1313.
- Osler, W.: Chronic Infectious Endocarditis, *Quart. Jour. Med.*, 1909, ii, 219.
- On the Visceral Complications of Erythema Exudativum Multiforme, *Am. Jour. Med. Sc.*, 1895, cx, 629.
- Patterson, J. D.: Pyorrhea Alveolaris, The Dental Summary, May, 1909.
- Pyorrhea Alveolaris, Johnson's Dentistry, ed. 3, 1915.
- Pierrepont, E. S.: Influence of Maternal Oral Sepsis on Fetus and Marasmic Children, *Lancet*, London, 1917, i, 837.
- Pirquet, C. E. von: Allergy, *Arch. Int. Med.*, 1911, vii, 259.
- Pottenger, F. M.: Anaphylaxis and Vegetative Nervous System, *New York Med. Jour.*, 1917, cvi, 293.
- The Antagonistic Action of the Vagus and Sympathetic Divisions of the Autonomic Nervous System in Pulmonary Tuberculosis, *Jour. Lab. and Clin. Med.*, 1916, i, 234.
- The Relationship Between the Nervous System and Therapeutics in Pulmonary Tuberculosis, *New York Med. Jour.*, 1916, civ, 939.
- The Syndrome of Toxemia an Expression of General Nervous Discharge Through the Sympathetic System, *Jour. A. M. A.*, 1916, lxvi, 84.
- Poynton, F. J., and Paine, A.: Further Contribution to the Study of Rheumatism; the Experimental Production of Appendicitis by the Intravenous Inoculation of the *Diplococcus*, *Lancet*, London, 1911, ii, 1189.
- Observations upon the Arthritis Produced in Rabbits by the Intravenous Inoculation of a *Diplococcus* Isolated from Cases of Rheumatism, *Tr. Path. Soc.*, London, 1900-01, lii, 248.
- Observations upon Certain Forms of Arthritis, *Brit. Med. Jour.*, 1902, ii, 1414.
- Remarks on the Infectious Nature of Rheumatic Fever, Illustrated by the Study of a Fatal Case, *Brit. Med. Jour.*, 1904, i, 1117.
- Some Further Investigations and Observations upon the Pathology of Rheumatic Fever, *Lancet*, London, 1910, i, 1524.

- The Etiology of Rheumatic Fever, *Lancet*, London, 1900, ii, 861.
- The Pathogenesis of Rheumatic Fever, *Path. Soc.*, London, 1900-01, lii, 10.
- The Relation of Malignant to Rheumatic Endocarditis, *Lancet*, London, 1902, i, 1036.
- Price, W. A.: Are Endamœbæ Important Factors in the Etiology of Pyorrhœa Alveolaris? *Jour. Nat. Dental Assn.*, 1915, ii, 143.
- Care of the Teeth, *Boston Med. and Surg. Jour.*, 1917, clxxvi, 317.
- Pathology of Dental Infections and Its Relation to General Diseases, *Dominion Dental Jour.*, 1916, xxviii, 121.
- The Dental Aspect of the Relation of Endamœba to Pyorrhœa Alveolaris, *Surg., Gynec. and Obst.*, 1916, xxii, 371.
- Price, W. A., and Bensing, L. P.: Are Endamœbæ Important Factors in the Etiology of Pyorrhœa Alveolaris, *Jour. Nat. Dental Assn.*, 1915, ii, 143.
- Rhein, M. L.: Deep-seated Alveolar Infections, *Surg., Gynec. and Obst.*, 1916, xxii, 33.
- Infected Areas Around the Ends of Roots of Teeth, *Jour. A. M. A.*, 1912, lix, 361.
- Scientific Treatment of Root Canals, *Dental Cosmos*, 1911, liii, 992.
- The Dental Aspect of Oral Infection, *Items of Interest*, 1914, xxxvi, 439.
- The Importance of Correct Differential Diagnosis of the Predisposing Causes in Cases of Interstitial Gingivitis or Pyorrhœa Alveolaris, *Jour. A. M. A.*, 1917, lxviii, 417.
- The Retention of Devitalized Teeth Without Danger of Focal Infection, *Jour. A. M. A.*, 1917, lxix, 474.
- Richet, C.: Les états. anaphylactiques en clinique mouvement med., 1914, ii, 1.
- Theories of Immunity and Anaphylaxis, *Tr. Internat. Cong. Med.*, 1913, Lect. IV, Bacteriol. and Immun., 13.
- Un nouveau type d'anaphylaxie, l'anaphylaxie indirecte; leucocytose et chloroforme, *Compt. rend. Acad. d. sc.*, 1914, clviii, 304.
- Richet, C., and Portier, P.: De L'action anaphylactique des certains venins, *Compt. rend. Soc. de biol.*, 1912, liv, 170.
- Robertson, R. S.: The Role of the Dentist in the Therapeutics of Internal Diseases, *New York Med. Jour.*, 1915, cii, 305.
- Roe, W. J.: Surgical Lesions Due to Oral Sepsis and Their Treatment, *Dental Cosmos*, 1915, lvii, 174.
- Rosenau, M. J.: General Principles of Preventive Medicine and Their Application to Dentistry, *Dental Summary*, 1917, xxxvii, 568.
- Rosenau, M. J., and Anderson, J. F.: A Study of the Cause of Sudden Death, Following Injection of Horse Serum, *U. S. Hygienic Laboratory Bull.*, 1906, No. 29.
- Further Studies Upon the Phenomenon of Anaphylaxis, 1909, *U. S. Hygienic Laboratory Bull.*, 1909, No. 50.
- Hypersusceptibility, *Jour. A. M. A.*, 1906, xlvii, 1007.
- Simultaneous Transmission of Resistance to Diphtheria Toxin and Hypersusceptibility to Horse Serum by the Female Guinea Pig to Her Young, *Jour. Med. Research*, 1906, x, 259.
- Studies on Hypersusceptibility and Immunity, *U. S. Hygienic Laboratory Bull.*, 1907, No. 36.
- The Specific Nature of Anaphylaxis, *Jour. Infect. Dis.*, 1907, iv, 552.
- Rosenow, E. C.: A Study of Pneumococci from Cases of Infectious Endocarditis, *Jour. Infect. Dis.*, 1910, vii, 411.
- Appendicitis and Parotitis, *Jour. Infect. Dis.*, 1916, xviii, 383.
- Bacterial Localization, *Jour. A. M. A.*, 1916, lxvii, 662.
- Bacteriology of Cholecystitis and Its Production by Injection of Streptococci, *Jour. A. M. A.*, 1914, lxiii, 1835.

- Causation of Gastric and Duodenal Ulcer by Streptococci, *Jour. Infect. Dis.*, 1916, xix, 333.
- Elective Localization of the Streptococcus from a Case of Pulpitis, Dental Neuritis and Myositis, *Internat. Jour. Orthodontia*, 1916, ii, 713.
- Elective Localization of the Streptococcus from a Case of Pulpitis, Dental Neuritis and Myositis, *Jour. Immunology*, 1916, i, 363.
- Etiology of Cholecystitis and Gallstones, *Jour. Infect. Dis.*, 1916, xix, 527.
- Herpes Zoster, *Jour. Infect. Dis.*, 1916, xviii, 477.
- Immunological and Experimental Studies on Pneumococcus and Streptococcus Endocarditis, (Chronic Septic Endocarditis), *Jour. Infect. Dis.*, 1909, vi, 245.
- Immunological Studies in Chronic Pneumococcus Endocarditis, *Jour. Infect. Dis.*, 1910, vii, 429.
- Iritis and Other Ocular Lesions on Intravenous Injection of Streptococci, *Jour. Infect. Dis.*, xvii, 403.
- Mouth Infections as a Source of Systemic Disease, *Jour. A. M. A.*, 1914, lxiii, 2026.
- Sore Throat, *Jour. A. M. A.*, 1917, lxviii, 1305.
- The Etiology of Acute Rheumatism, Articular and Muscular, *Jour. Infect. Dis.*, 1914, xiv, 61.
- The Bacteriology of Appendicitis and Its Production by Intravenous Injection of Streptococci and Colon Bacilli, *Jour. Infect. Dis.*, 1915, xvi, 240.
- The Etiology and Experimental Production of Erythema Nodosum, *Jour. Infect. Dis.*, 1915, xvi, 367.
- The Newer Bacteriology of Various Infections as Determined by Special Methods, *Jour. A. M. A.*, 1914, lxiii, 903.
- The Relation of Dental Infection to Systemic Disease, *Dental Cosmos*, 1917, lix, 485.
- Transmutation Within the Streptococcus-Pneumococcus Group, *Jour. Infect. Dis.*, 1914, xiv, 1.
- Rosenow, E. C., and Offerdal, S.: The Etiology and Experimental Production of Herpes Zoster, *Jour. A. M. A.*, 1915, lxiv, 1968.
- Rosenow, E. C., and Sanford, A. H.: The Bacteriology of Ulcer of the Stomach and Duodenum in Man, *Jour. Infect. Dis.*, 1915, xvii, 219.
- Sanford, A. H.: The Relation of Amœbrasis to Pyorrhœa Alveolaris, *Surg., Gynec. and Obst.*, 1916, xxii, 27.
- Shivdas, S. P.: Role of Pyorrhœa Alveolaris in Causation of Chronic Bronchitis and Asthma, *Practitioner*, London, 1916, cvi, 428.
- Schultz, F.: Physiological Studies in Anaphylaxis. The Reaction of Smooth Muscle of the Guinea Pig Sensitized with Horse Serum, *Jour. Pharmacol. and Exper. Therap.*, 1909, i, 540.
- Smith, A. J., and Barrett, M. J.: Emetin in the Treatment of Peridental Suppurations, *Dental Cosmos*, 1915, lvii, 1201.
- The Parasite of Oral Endamœbrasis, Endamœbæ Gingivitis. (Gros), *Jour. Parasital*, 1915, i, 159.
- Steinharter, E. C.: Acute Arthritis Experimentally Produced by Intravenous Injection of the Staphylococcus Pyogenes, *Boston Med. and Surg. Jour.*, 1916, clxxv, 59.
- Gastric Ulcer Experimentally Produced, *Boston Med. and Surg. Jour.*, 1916, clxxiv, 678.
- Streitmann, W. H.: Oral Sepsis as Related to Systemic Disease, *Items of Interest*, xxxvii, 930.
- Thoma, K. H.: Diagnosis and Treatment of Alveolar Abscesses Caused by the Diseases of the Dental Pulp Based on Pathological and Radiographic Study, *Items of Interest*, 1917, xxxix, 516.
- Oral Abscesses, Boston, Ritter and Co., 1916.

- Tousey, S.: Dental Infections in Systemic Disorders, New York Med. Jour., 1916, civ, 1269.
- Trudeau, E. L.: Two Experiments in Artificial Immunity Against Tuberculosis, Med. News, 1905, lxxxvii, 633.
- Ulrich, H. L.: The Blind Dental Abscess, Boston Med. and Surg. Jour., 1916, clxxiv, 169.
- Vaughan, V. C.: Infection and Immunity, Jour. Mich. Med. Soc., 1916, xv, 8.
- Poisonous Proteins, Jour. Lab. and Clin. Med., 1916, i, 631, 851.
- Protein Poison, Jour. A. M. A., 1916, lxxvii, 1559.
- Protein Split Products in Relation to Immunity and Disease, Philadelphia, Lea and Febiger, 1913.
- Vaughan, V. C., and Wheeler, S. M.: The Effects of Egg White and Its Split Products on Animals: A Study of Susceptibility and Immunity, Jour. Infect. Dis., 1907, iv, 476.
- Webb, G. B., and Williams, W. W.: Immunity in Tuberculosis, Jour. Med. Research, 1911, n. s., xix, 1; Jour. A. M. A., 1911, lvii, 1431.
- Weil, R.: Anaphylaxis and Its Relation to Problems of Human Disease, Lancet-Clinic, 1913, cx, 568.
- Further Studies of Serum Sickness, Proc. Soc. Exper. Biol. and Med., 1916, xiv, 60.
- Williams, A. W., and Sholly, A. I. von, (et al): Significance and Prevention of Amebic Infections in the Mouths of Children, Collect. Stud. Research Lab., Dept. Health, N. Y., 1916, viii, 405.
- Winternitz, M. C., and Quinby, W. C.: Experimental Nephropathy in the Dog. Lesions Produced by Infection of the Bacillus Bronchisepticus Into the Renal Artery, Jour. Urology, 1917, i, 139.
- Wright, W. M.: Some Pathologic Conditions of the Mouth of Interest to the Physician and Dentist, Penn. Med. Jour., 1916, xx, 105.
- Young, J. H.: Tonsillectomy as a Therapeutic Measure in the Treatment of Chorea and Endocarditis, Boston Med. and Surg. Jour., 1915, clxxiii, 356.
- Zinsser, H., and Parker, J. T.: Studies on Bacterial Anaphylaxis and Infection, Jour. Exper. Med., 1917, xxvi, 411.

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